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THE METHODS AND MANNERS OF GREAT MEN OF MEDICINE

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When I think of doctors and their work, I am reminded of the oft repeated but nevertheless significant quotation: "There are men and classes of men that stand above the common herd; the soldier, the sailor and the shepherd not infrequently; the artist rarely; rarelier still, the clergyman; the physician almost as a rule. . . ." Perhaps Robert Louis Stevenson, when he wrote these lines, had in mind men of the calibre that constitute the membership of this great organization.

Let us look for the reasons why the physician stands high in the professional world. First of all, as we are well aware, is the prolonged formal education, followed by years of postgraduate work. During and following this period he deepens his habit channels; disciplines his mind; and increases his logic of thinking by the constant application and reapplication of information for useful purposes. Then too, the work hours are long! His work day is not three or four hours, but twelve to fourteen hours, and more if need be, seven days a week, eleven to twelve months of the year. From the standpoint of association, he is thrown with men of equal intellectual attainments from whom he gains the wisdom to recognize different viewpoints, and from his older confrères he receives the inspiration to set into motion his own creative thinking and under their tutelage to use his technical skill. In addition to the long hours of reading and studying, which must continue, tremendous responsibility tests his judgment, matures his thinking. But he truly stands above the herd when he finds there is a human element in the practice of medicine. This he discovers when listening to his patients' trials and tribulations; their joys and sorrows; seeing their smiles

Presidential address delivered during the annual assembly of The Southwestern Surgical Congress, Salt Lake City, Utah, September 21–23, 1953.

and tears; hearing of their attainments; of the attainments of their children and their childrens' children; or perhaps hearing the frustration of one gone wrong. From each patient with whom he talks he gathers a sprinkle of philosophy which must fall upon him—even though ever so gently. Each one has a share of philosophy—the rich and the poor, the educated and the uneducated—and not infrequently among the latter will be found occasionally the most stately tree of the forest. The rugged edge of one's personality is smoothed away by such contacts.

So much for the human equation. Now! On the scientific side, he takes great pride in the prevention of certain diseases; in controlling metabolic disorders and in improving or curing great lists of diseases with medical or surgical means. He mends broken bodies and extirpates tumors with success. Truly he can say "I was for that time lifted above the earth, and possessed joys not promised in my birth." But there come times in every physician's life when the proper diagnosis eludes him; when the manufacturing centers of the body go off on a slow-down strike in spite of urgings; and the down-stream of health flows right on. Too, there comes a time when one must helplessly observe the withered body of cancer until the last vestige of life departs from it. These reactions in a physician's life "sharpen the fine edge of sympathy." These are things that should make physicians great—the things that should make them appear as glistening peaks above the crowd.

I have just mentioned some of the factors that may make physicians stand head and shoulders above the crowd. However, I wish particularly to discuss the qualities, the methods, the arts, the philosophies which have been characteristic of a few of the great masters of medicine who have stood high in history above their medical confrères. I shall avoid dealing in personalities, insofar as possible, utilizing only the system, the conduct, the philosophy or ethics which may be common to each of these great men, or to them as a group.

These we shall consider under three major headings: "Method," "Art of Medicine," and "Faith."

Method

Now we come to a consideration of method.

Method is the apportioning of one's irreplacable time for various purposes, so that the hours of the day and the months of a life may be spent efficiently to obtain and impart knowledge. It is the careful planning of a course. It is the recognition of important steps which must be taken to arrive at a goal. Only a few of the more important steps will be discussed here.

Observation: This leads us to a thought on observation.

Early in life some people have an intense interest in the things about them. This brings out one of the characteristics that have made men great, and that is *Observation*. Philosophy, Plato tells us, begins with wonder, and the first step is observation of nature. This is true in medicine. It is no surprise then to find that these great men started making original observations early in life. Contrary to the present day tendency to specialization, their observations were made in

many different fields of endeavor. The great engineer and sculptor, Leonardo Da Vinci, with his airplanes, his paintings and his drawings of anatomy is an example. To be a good observer one must "see" as well as "look." He must have an alert, receptive, and a disciplined mind. He must have the power to concentrate and the ability to describe. When Archimedes lowered himself into his bath and the water overflowed, had he not had an alert, receptive and thinking mind the science of hydrostatics may have been delayed. As Sir Isaac Newton drove along the highway and saw an apple fall and be pulled to the earth by gravity, had he not had an alert, receptive mind with keen observation, the knowledge of the force that holds the universe together might not yet be solved. Had the Stophylococcus culture not been contaminated with the spore of *penicillium notatum*, and if the bacterial properties had not been noted and fully recorded by Sir Alexander Fleming, penicillin and other antibiotics may not even yet have been made available to cure the ill. It is with reason that Pasteur said "In the fields of Observation chance favors only the mind which is prepared."

It is not enough just to observe; to see. One must record his observations and accuracy is the keystone of description, for however well the memory may be developed, the record should be made at once. Carry a notebook, a scratch pad, or make notes on the edges of the page as you read. This was a habit of Osler's. Sometimes a thought, an idea, is fleeting, like the light of a shooting star. To be retained it has to be recorded. Now, John Foster has aptly expressed the value of recording one's experiences:

"An observant man, in all his intercourse with society and the world, carries a pencil constantly in his hand, and, unperceived, marks on every person and thing the figure expressive of its value, and therefore instantly on meeting that person or thing again, knows what kind and degree of attention to give it. This is to make *something* of experience."

Experiment: Following closely on the heels of observation comes experimentation. Research always has its beginning in ideas. The development of research is carried out through work. Work entails reading, experimentation, recording, tabulating. Research work may be carried out successfully in either a general or specialty practice. Some of the profound discoveries were made by men who were not associated in teaching centers. Well known examples are Jenner and his cowpox vaccination, and Beaumont and gastric secretions. A well known discovery by a less well known scientist is one by Doctor George Oliver, of Harrogate, who spent his leisure winter hours in experiments. He had devised an instrument for measuring the thickness of the radial artery at the wrist. One evening he gave his young son an injection of an extract made from the suprarenal gland prepared from material furnished by the local butcher. After the injection he noted a rather sudden and marked change in the thickness of the artery. With enthusiasm he took his experiment and the extract to Doctor Schafer, a physiologist in London, who at the moment was carrying out an experiment on the blood pressure of a dog. Doctor Schafer was annoyed at the interruption of this country physician. However, after his own experiment of

recording the dog's pressure was finished, to show Oliver his nonsense and to get rid of him, he injected some of Oliver's extract into the same dog on which he had just completed the blood pressure recording, and Schafer stood amazed to see the mercury mounting in the arterial manometer until the recording float was lifted almost out of the distal limb. The original experiment with adrenalin was thus performed by a country physician. Banting, too, developed his idea of insulin while in practice and later brought it to fruition in Toronto. Great men of medicine, with the possible exception of the Greeks, have been given to experiment and research. With discovery comes great joy and pride.

Persistence: No idea, however important, no ambition, however strong, without force persistently applied can be brought to fruition. Persistence is an essential quality in the successful accomplishment of almost any endeavor. It is especially true in medicine. Day after day one must pursue his subject until it is accomplished. There is small room for the dreamer, who has the jelly of ambition but not the bony framework. There is small room for the fellow who says it should be thusly done, but never puts a hand to it to accomplish the feat. There is no place for the fellow who carouses at night and lies abed late in the morning. Persistence is a requirement that is not listed in the catalogue, but it is an essential nevertheless. Big men of medicine usually develop it early in life.

For instance, a young woman, while in labor, died suddenly. Her attending physician was young—Doctor James McKenzie. This was the beginning of his determination to investigate heart disease—the very beginning of his "Wait and See" philosophy. He devised an instrument for making tracings of the pulse, the pulsations in the neck, and the relationship of these to the heart beat in health and in disease. He made thousands of tracings; took careful histories; did careful examinations; but above all, he followed the patients and their activities for many years, or until death. Each year he would classify the tracings of these patients according to their health or to their illness. There would be literally thousands of tracings to be carefully examined. The work of classifying them with the clinical picture went on far into the night and on Sundays. It was a task so great that lesser men would have given it up long before completion—but not James McKenzie. He continued through the years to make these careful studies in his own private practice in Brumley until he himself was sure of the meaning of these tracings. Then he gave his epoch-making findings on the heart to the world. This is a truly great example of persistence and determination.

Weir Mitchell began his notebook descriptions of nature landscapes early in boyhood. Wherever he went he carried a notebook and pencil. He had the persistence to make his descriptions each time he went afield. The thoroughness of his method and his persistence were no small factors in his success as a great physician.

Imparting of Knowledge: When nature has been kind enough to give up her secrets, it has been characteristic of great men to have an intense desire—a persistent desire—to give the knowledge to confrères and to the world. "Observe,

record, tabulate, communicate" is the way Thayer described the manner of Osler in driving home his views. To communicate his knowledge apparently was one of his great pleasures, because he used letters, papers and addresses before organizations to give the information he had gained to his fellowmen. He entertained in his home, students and staff alike, and much of the time was given to the dispensation of medical knowledge, always with wit, always with shafts of enthusiasm.

Hippocrates, Galen, Vesalius, Paré—to mention the better known names of the more ancient part of the history of medicine—wrote profusely and were inspired to start schools and to teach the art and the science of medicine to their younger fellowmen.

The work of Sir James McKenzie has been mentioned before. In addition to the persistence in carrying out his experiments, he had an intense desire to give this valuable information to the world. To do this, he gave up a lucrative practice in his hometown and at a great financial risk to himself, moved to London in order that he might better be able to bring his findings to the medical world.

The Mayo Brothers did more than establish the first great private institution for the care of the ill. They made provisions for carrying on experimental and clinical research. The information which they discovered was freely given to visiting physicians and to the world through their participation in medical meetings and through their writings.

All the great names in the more modern medical history are too numerous to mention. However, they too have undertaken to teach, to write, and to impart their knowledge unreservedly to their confrères. Perhaps we know these men only by their writing, and so it is even today as we sit with our fellow physicians it will be "writings" which will separate and classify those of us who are to become known to posterity.

This concludes our consideration of "Method".

Art of Medicine

We are told that the strict application of just science to patients brings only a measure of success. To it must be added the "Art" of Medicine.

Of what does the "Art" of Medicine consist? And what did these men have to make them great in the "Art" of Medicine? This is what they had: sincerity of purpose, wit when needed, honesty without fear, integrity to give confidence to the patient, and an insatiable desire to inspire them. They also had love for people as a common denominator; firmness and gentleness properly applied; sympathy in the hour of need; and finally understanding, equanimity, imperturbability, and humility, of which four I shall say more.

Understanding: One may listen without hearing, or hear without understanding, as well as look without seeing. It is not always easy to want to understand the problems of someone, even though a patient. But the physician who has understanding and who exercises this rare talent soon becomes a pillar of strength; a ray of hope; a friendly peaceful pool in which to pour trouble; a catalyst to

inactivate disturbing problems on the one hand, and to fortify the pleasures of life on the other, or he becomes a force to augment the patient's courage to endure his lot stoically.

In "The Road to Bithynia" by Doctor Frank E. Slaughter, it is stated that Saint Luke knew that the highest duty of a physician is to receive the troubled thoughts of those sick in mind and body and to give to them the confidence and the assurance they so badly need.

When one manifests his sincere interest in this manner, that is *Understanding and Humanism.*

Equanimity and Imperturbability: To this understanding and humanism must be added equanimity and imperturbability. These fit together like a hand in a glove. Equanimity is that inward mental state of calmness and understanding, fluctuating only very slightly with circumstances.

Perturbability is the outward bodily manifestation of the unsettled mind. Imperturbability means coolness of mind; ability to follow an even course when everything is going wrong; the outward show of unfrustrated nerves under the most trying situations. It does not mean that a person with imperturbability is phlegmatic, cold or indifferent. Rather it means that in spite of a highly sensitive nature he remains serene and unaffected by disturbing conditions.

It is said that Osler was severely criticized as being cold and unsympathetic because once after leaving the room of a desperately ill child he whistled as he went down the corridor. He did whistle—to keep from crying!

The great men had much equanimity and imperturbability.

Humility: One of the greatest virtues of the great men of medicine has been that they have had great humility.

During the celebration of the eightieth birthday of William H. Welch, dignitaries throughout the world, including the President of the United States, heaped deserving praise upon him. In his reply he brushed aside their attempts at tribute by calling attention to the fact that so much more remains to be done than has been accomplished; that problems awaiting solutions are so numerous and urgent, that the need to get knowledge so imperative that one's mental attitude should be far removed from satisfaction. Osler speaking to students on the grace of humility said: "So should you now at the onset of your journey take the reed of humility in your hands in token that you appreciate the length of the way, the difficulties to be overcome, and the fallability of the faculties upon which you depend."

As one reads about great things and about great discoveries, and about great men, there is one very important outstanding impression—that gifts of great attainment come from on High.

Faith

Great men of medicine have had an abiding faith in a higher power. Many have had rather deep religious beliefs. A few have written extensively about their convictions—among them Osler, Pasteur, and Sir Thomas Browne. Probably the best known work is Religio Medici by Sir Thomas Browne, a book which can be

read and lived by. Doctors in general may well express their creed as did a Hebrew author, who wrote before the time of Saint Luke:

The Prayer of a Physician

" O stand by me, my God, in this truly important task;
Grant me success! For—
Without Thy loving counsel and support,
Men can avail but naught.
Inspire me with true love for this my art
And for Thy creatures,
O grant—
That neither greed nor gain, nor thirst for fame, nor vain ambition,
May interfere with my activity.
For these I know are enemies of Truth and Love of men,
And might beguile one in profession
From furthering the welfare of Thy creatures.
O strengthen me.
Grant energy unto both body and soul
That I might e'er unhindered ready be,
To mitigate the woes,
Sustain and help
The rich and poor, the good and bad, enemy and friend.
O let me e'er behold in the afflicted and suffering,
Only the human being."

PRECAUTIONS IN THE TREATMENT OF STRICTURES OF THE COMMON DUCT

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In any discussion of the treatment of strictures of the common duct we should bear in mind the fact that operative trauma is the cause of at least two-thirds of the strictures. Inflammation involving the common duct accounts for about 20 per cent of the strictures; the causation of many of these is no doubt related to trauma. Chronic pancreatitis has been responsible for 8.6 per cent of the strictures observed in our clinic. Almost invariably the operation during which trauma is sustained is cholecystectomy. Rarely indeed does a stricture develop following cholecystostomy or choledochostomy.

A fairly large number of patients gave a history of development of jaundice three to six months following cholecystectomy. Even in patients having a history of delayed development of jaundice trauma still may be the causative factor. For example, a suture may have been placed through part of the wall of the common duct and as time progressed, inflammation finally invaded the duct sufficiently to cause complete fibrosis. On other occasions a small abscess may have formed just anterior to the common duct, or a collection of bile may have developed in this area. Although the latter two causes are not specifically related to technical error, they still are related to the operation (fig. 1).

PRECAUTIONS IN THE PREVENTION OF STRICTURES

1. *Obtain Adequate Exposure of Operative Field:* In biliary surgery adequate exposure is very important in the prevention of technical errors. Exposure may be obtained by a transverse or a longitudal incision; the author prefers the latter, because it appears to him to allow more satisfactory vision of the operative field.

2. *Adequate Time Needed for Dissection and Identification of Structures:* Since technical errors in dissection and operative technic during cholecystectomy may be made so easily, it is obvious that extreme care must be maintained at all times during the operation. This meticulous care is probably more important during cholecystectomy than choledochostomy, because in the latter operation the common duct as well as the cystic duct must be visualized before the operation has progressed very far. Accordingly, there is no justification for hurrying while cholecystectomy is being done.

3. *Be Aware of Congenital Malformations:* In no area of the body are congenital malformations more common than in the biliary ducts and blood vessels leading to the liver. Accordingly, one can not perform operative maneuvers on the basis of known locations of various anatomic structures. A surgeon must visualize the
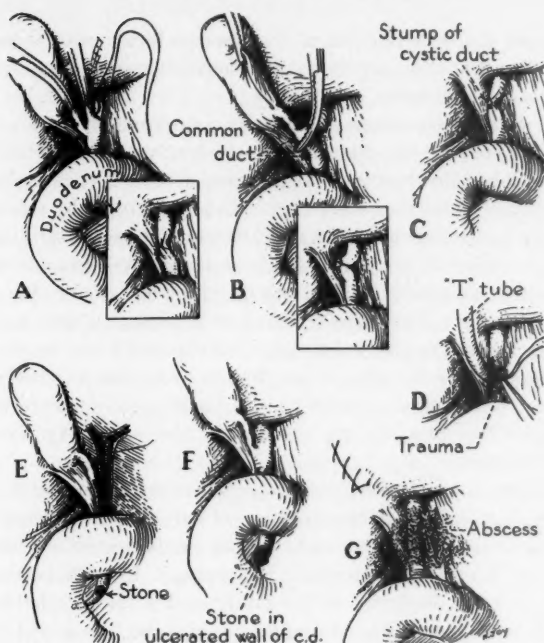
FIG. 1. Mechanisms in production of stricture of common duct; (*A*) transfixion with a needle; (*B*) ligation with the cystic duct; (*C*) ligation of the cystic duct too close to the common duct; (*D*) chronic diffuse sclerosing pancreatitis; (*E*) cholangitis; (*F*) ulceration of of the wall by stone; and (*G*) abscess or local collection of bile. (From Cole in *Operative Technic*, Appleton-Century-Crofts.)

various anatomic structures and protect against damage to them in every individual case. Common anomalies are accessory hepatic ducts, accessory cystic arteries, and right hepatic artery anterior to the common duct. Often Hartmann's pouch is large and lies against the common duct. If inflammation is present it may be difficult to dissect Hartmann's pouch from the common duct without damaging the latter structure.

4. *Cut No Structure Until It is Definitely Indentified:* Since anatomic anomalies are seen so commonly in this area, and since the structures are so often obscured by inflammation it becomes exceedingly important to identify all structures before they are cut.

5. *The Junction of the Cystic and Common Ducts Must be Exposed During Cholecystectomy:* The initial procedure practiced by most surgeons in a cholecystectomy is to start dissection near the junction of the cystic and common ducts. Often the cystic duct is buried in adhesions, on which occasion it is usually desirable to incise the peritoneum over the common duct and expose this structure; this makes dissection of the cystic duct much safer.

6. *The Cystic Artery and Duct Should be Ligated Separately:* Ligation of the duct and artery in one ligature results in strangulation of a large mass of tissue

which encourages the development of an abscess. Ligation of these two structures individually, likewise allows better identification of them and prevents damage to adjacent structures.

7. *Control Arterial Hemorrhage by Pressure and Accurate Ligation of Bleeding Point:* One of the common mechanisms by which trauma is inflicted on the common duct is to clamp the duct while controlling accidental hemorrhage from the cystic artery. When hemorrhage develops in the operative field, the surgeon must not stab blindly in the region of the bleeding point, because the usual source of bleeding is the cystic artery, which tends to retract posterior to the common duct when severed. To blindly control this bleeding, one would obviously have to clamp the common duct. The correct procedure is to place a stick sponge over the bleeding point and insert the index finger of the left hand in the foramen of Winslow. The right hepatic artery can then be compressed between the index finger and the thumb, thus controlling the bleeding. By slowly releasing this compression, the bleeding can be visualized, and an artery forceps applied accurately to the vessel.

8. *When Adhesions are Dense, Start Dissection at the Fundus of the Gallbladder:* Now and then, particularly in the presence of subacute cholecystitis, adhesions will be so dense around the cystic and common ducts that dissection in this area is entirely unsafe. To prevent damage to the common duct it is better, under such circumstances, to start dissection at the fundic end of the gallbladder. It usually is preferable to open the gallbladder and evacuate the bile and stones. One can then insert a finger into the gallbladder and thus obtain assistance in dissection at the lower end.

9. *Remember that 65 to 75 Per Cent of Strictures of the Common Duct Result from Trauma:* In about 65 per cent of cases the etiology of strictures positively can be traced to trauma. In another 15 or 20 per cent trauma or error in operative technic may be related to causation.

10. *Miscellaneous Precautions:* Numerous other precautions should be taken during cholecystectomy. The most important of these is accurate differentiation between the cystic artery and right hepatic artery. In 30 to 35 per cent of the cases, the right hepatic artery projects forward after it emerges from beneath the common duct. Time and time again the author has caught himself in the process of placing a clamp on the right hepatic artery; fortunately he has always taken another look and thus made the correct identification. This error of ligating the right hepatic artery instead of the cystic would not lead to stricture, but might lead to death of the patient because of hepatic necrosis.

PRECAUTIONS AND INDICATIONS IN OPERATIONS FOR STRICTURE OF THE COMMON DUCT

1. *Establishment of the Diagnosis:* If the common duct is completely occluded by a ligature, jaundice will develop in 36 to 48 hours. If the duct is cut and not ligated, a bile fistula will develop through the wound if the region of the duct has been drained properly; if the peritoneal cavity has not been drained, bile peritonitis will develop. When an obstruction of the duct is suspected after cholecys-

tectomy, often it is desirable to give an enema and note the color of the stool—it must be remembered that the first stool after operation may contain bile which entered the intestinal tract before the operation. Likewise if the obstruction is partial, it may be difficult to identify since some bile may get through and produce a normal colored stool. Jaundice may be delayed up to six months because of several factors. One of these factors may be partial obstruction of the common duct by ligature, and development of inflammation and subsequent fibrosis of the duct. Reference already has been made to the collection of pus and bile anterior to the common duct as a possible cause of stricture.

The diagnosis of chronic stricture may be difficult, particularly since the symptoms are so variable. At the onset of the jaundice, pain may not be present, but as stated above, it develops later, probably being related to development of infection. One must remember that completeness of a stricture varies from week to week until the fibrosis becomes complete. Variation in the degree of obstruction is dependent upon the severity of the infection and inflammation. As the obstruction varies, so does the jaundice and color of the stool. Chills and fever secondary to suppurative cholangitis usually develop; likewise diarrhea and loss of weight usually take place. However in many individuals with complete obstruction of the common duct, the mucosa of the intestinal tract develops the ability to excrete bilirubin, thus giving rise to colored stools.

2. *Preoperative Care and Preparation of the Patient for Operation Must be Meticulous:* Since these patients commonly have had two or three previous operations and are suffering from malnutrition, we must be particularly cautious, and prepare them for the operations which are long and hard on the patients. Obstruction of the common duct gives rise to biliary cirrhosis. Sooner or later cellular necrosis takes place. When this occurs, serious hepatic damage develops with obvious consequences. Particularly related to the hepatic damage are hypoproteinemia and hypoprothrombinemia. Most of these patients are particularly susceptible to electrolyte deficiency, particularly potassium. Deficiency of all vitamins related to dietary intake is to be expected since the appetite is poor. Accordingly, blood and plasma must be given freely to correct the anemia and hypoproteinemia. Electrolyte levels must be watched closely particularly when a biliary fistula is present. A vitamin K like product should be given the day before the operation and continued for 10 days thereafter.

3. *Choose the Correct Time for Operation:* If jaundice or a biliary fistula develops two or three days after cholecystectomy, trauma to the common duct should be suspected until proved otherwise. Other causes of biliary fistula are slippage of a ligature from the cystic duct or severance of an accessory hepatic duct without its ligation. In either of these conditions bile will be present in the stool. If jaundice develops early after operation there are relatively few explanations other than trauma which usually consists of ligation or excision of a part of the duct. In general, the sooner after operation the repair is made, the more successful the repair will be.

In case a biliary fistula is present, there is indication to operate as soon as possible, before the fistula closes and perhaps completely obscures the location

of the intrahepatic duct. When a fistula is present it is very easy to insert a probe and follow the tract down to the hilus of the liver and thus identify the intrahepatic duct.

Adhesions are most dense following trauma to the common duct between the fourth and twelfth week after operation particularly if there has been leakage of bile after the initial operation. This factor may slightly influence the choice of time for operation, but in general there is little justification for much delay.

4. *Choose the Operation which Best Meets the Needs as Influenced by Anatomic Findings:* A surgeon must look for both ends of the common duct, but naturally he looks for the proximal end first since nothing can be done with the distal end alone.

If both ends of the common duct are found, usually it is possible to do an end to end anastomosis. The author has not been very successful in splitting the head of the pancreas posteriorly, after the method of Cattel,[1] and finding the distal end. After looking unsuccessfully for 30 to 40 minutes for the distal end, usually we resort to construction of a Roux Y arm of jejunum to replace the lost common duct (fig. 2).

When the proximal duct is found, and the distal end not available, one of two procedures must be performed, (1) construction of a Roux Y arm of jejunum, or (2) anastomosis of the hilar duct to the duodenum. The author has had much better results with the use of the Roux Y arm, but Walters[5] reports good results with anastomosis of the duct to the duodenum. If the duct is buried $\frac{1}{2}$ inch or
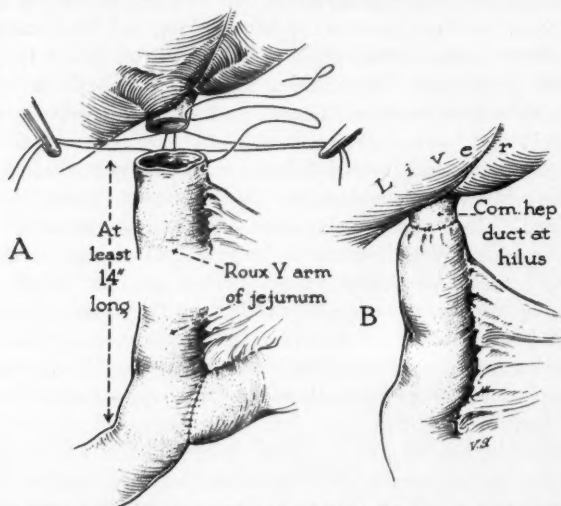
Fig. 2. Repair of stricture by anastomosing the hilar duct to the end of a Roux Y arm of jejunum when the distal end of the duct cannot be found. (*A*) interrupted mattress sutures of fine silk are taken through the common duct and jejunum as illustrated. (*B*) Appearance of the suture line after all sutures are tied. The hilar duct is not always as large as shown. When it is smaller a smooth junction will not be achieved so readily. When the hilar duct is large and relatively free from inflammation we do not use a T tube or catheter for a prosthesis. (After Cole and associates in Ann. Surg.)

less in the hilar area of the liver it can be exposed by excision of liver tissue, and insertion of the end of the Roux Y arm of jejunum up to meet the mucosa of the duct. If the opening is small it may be necessary to excise the musculature of the jejunum so that the mucosa and submucosa of the jejunum may be inserted into the small opening in the liver to meet the mucosa of the bile duct.

On a few occasions the author has been unable to find a remnant of intra-hepatic duct. Under such circumstances he adopts the technic of aspirating the liver at the hilus until a duct is found. The liver then is excavated, being careful to feel ones way with an aspirating needle to prevent severance of a major vessel. Often indeed, a major vessel blocks our excavation down to the duct which may be as far as $1\frac{1}{2}$ inches beneath the surface of the liver. However, numerous aspirations of the duct through this area invariably result in production of a fistula two or three days after operation. We have attached the end of a Roux Y arm of jejunum to this opening in the liver on four or five occasions, and have had surprisingly good results in two or three.

5. *Choose the Correct Prosthesis:* Actually this precaution is somewhat para-doxical in so far as we do not yet have the perfect prosthesis. We have abandoned the use of the vitallium tube because it tends to plug as do all other tubes. At the present time we prefer to use a T tube as illustrated in figure 3; one arm of this T tube is inserted upward into the intrahepatic duct. The largest possible tube is chosen to maintain a large opening. The tube usually is split so that part of the tube will go up the right duct and the rest of the tube will go up the left duct. We have used the McArthur method quite successfully on certain occasions. By this procedure, a rubber tube 3 or 4 inches long is inserted; the tube is held in place by sutures leading to the outside anchored over a rubber tube or button. By cutting this suture the rubber tube will pass on into the gastrointestinal tract, but only if a Roux Y arm of jejunum is constructed in place of the distal end of the common duct.

There is considerable difference of opinion as to how long a prosthesis should be left in place. This difference of opinion is based on two factors, (1) that a stomal opening needs support during healing in the presence of inflammation and, (2) that any prothesis will produce undesirable foreign body reaction. Lary and Scheibe[3] have reported experiments showing that when end to end anasto-mosis of the common duct is made in dogs, less fibrosis and less stricture develops if no prosthesis (rubber tube) is left in place. These authors produced dilatation of the common duct by ligature which was removed one week later, the duct transected, and an anastomosis made between the two ends. In 11 of 12 animals having the suture line supported with a T tube, a stricture developed, whereas in only 3 of 16 animals in which the anastomosis was made without support by a rubber tube, did a stricture develop. In all animals having drainage of the com-mon duct with a T tube culture of the bile was positive, whereas culture was positive in only 56 per cent of animals not having a T tube inserted for support of the suture line. In similar experiments Harridge[2] noted that decompression proximal to an anastomosed common duct of dogs tends to increase stricture formation. In animals having decompression with a T tube for 24 to 81 days,
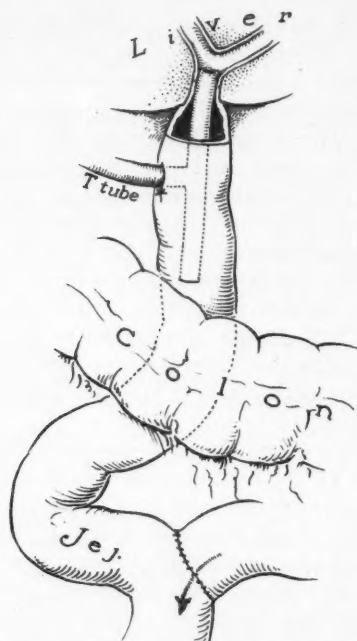
FIG. 3. When the hilar duct is small or its wall much inflamed, or when liver tissue must be penetrated to reach the duct we believe a prosthesis is desirable. At present we are using a T tube for this purpose particularly since the long arm through the jejunum tends to hold the tube in place. When the bifurcation of the ducts is near the suture line, the end of the tube should be split, and a half inserted up into each duct. (After Cole and associates in Ann. Surg.)

stricture was noted in 42.8 per cent; in animals having short term decompression for an average of seven days a stricture developed in 21.4 per cent, whereas in animals having no decompression, there were no strictures. The author considers these experiments very important in relation to clinical practice; however, it is possible that the findings cannot be translated literally to clinical experience, because the experiments on animals were made in the absence of infection. Accordingly, the author is of the opinion that in the absence of significant inflammatory changes in the human case, no prothesis should be used. However, it appears logical that in the presence of a thickened and inflamed duct wall a prosthesis should be used— for 8 to 10 months—particulary if the lumen of the duct is small.

DISCUSSION

The role of trauma in production of strictures of the common duct, and precautions during cholecystectomy have been emphasized. One of the additional features which I wish to stress in this presentation is the difficulty in determining—in the chronic cases—when jaundice is produced by hepatic

insufficiency and when it is produced by common duct obstruction. When jaundice is produced by suppurative cholangitis secondary to obstruction of the common duct, chills and fever—with intermittent acholic stools—are almost universal as the infection and obstruction vary in intensity. When jaundice is produced by hepatic insufficiency chills and fever may be absent although prolonged and neglected suppurative cholangitis invariably results in hepatic insufficiency. However, in severe insufficiency the stools usually are acholic, thus presenting a clinical picture similar to obstruction of the duct. This hepatic insufficiency, often associated with cellular necrosis, may or may not be associated with a severe biliary cirrhosis. We recently have made an erroneous diagnosis of recurrence of a stricture following repair, in 2 patients who actually had such severe hepatic damage that stools were acholic and a fatal outcome resulted. Unfortunately we reoperated upon one of these patients, using a Longmire[4] procedure, without inspecting the stoma between the hilar duct and Roux Y arm of jejunum. As indicated in the protocols she developed numerous complications which no doubt were partially responsible for her death. At autopsy, the stoma between the hilar duct and arm of jejunum was wide open (fig. 4).

Accordingly, it is very essential that the patient be assayed carefully from the standpoint of liver function, since it is possible that jaundice and acholic stools can be produced by biliary cirrhosis and cellular damage. Liver function tests and color of the stool will be extremely helpful in avoiding this error. If liver function tests are positive and the stools are cholic, there is a strong possibility that the jaundice may be caused by hepatic insufficiency. We emphasize again that hepatic insufficiency may produce jaundice without acholic stools; as the insufficiency increases, stools will become acholic.

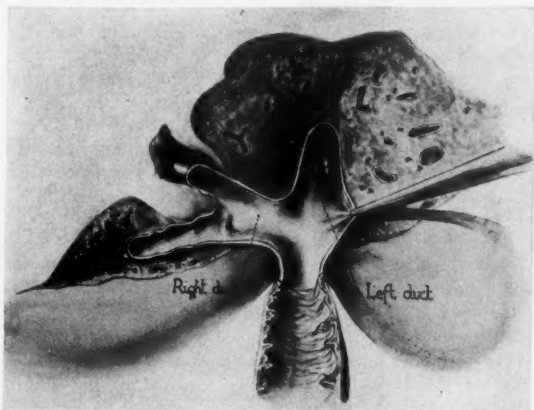FIG. 4. Artist's drawing of the stoma (at autopsy) between the hilar duct and Roux Y arm of jejunum in case 1. The patient had jaundice and intermittent acholic stools, but autopsy revealed no obstruction. Severe hepatic damage was the explanation of the manifestations just mentioned, as well as terminal gastrointestinal hemorrhage. An organized blood clot is attached to the wall of the left duct.

Because of the tendency for cirrhosis and severe hepatic insufficiency to develop in patients with strictures of the common duct one should not delay repair very long because the time required for development of severe hepatic damage differs in all patients. We have adopted the principle of watching the liver function tests very closely while observing a patient with occasional attacks of jaundice and acholic stool. If we think there is perhaps a chance that the obstructive attacks are becoming less frequent and less severe, we like to delay operation. However, if liver function tests become positive during this period of observation, delay may then be unsafe because severe damage may result from hepatic insufficiency.

Sometimes portal hypertension with production of bleeding esophageal varices develops in patients with common duct obstruction. On certain occasions after the active bleeding is controlled, it may be desirable to do a portacaval or splenorenal shunt.

CASE REPORTS (ILLUSTRATING DIFFICULTY IN DIFFERENTIATING SEVERE HEPATIC INSUFFICIENCY FROM COMMON DUCT OBSTRUCTION)

Case 1. G. T., a white woman, aged 57, had a cholecystectomy and choledochostomy done elsewhere in April 1947. Stones were found in the common duct. The stones were removed, and a T tube was inserted for drainage. Convalescence was stormy and six weeks after operation she became jaundiced. The jaundice disappeared and recurred intermittently.

She was admitted to The Illinois Research Hospital April 20, 1948. At this particular time there was no jaundice. Liver funtion tests were normal. At operation April 24, 1948 a stricture of the common duct was found. The stricture was located just distal to the bifurcation of the ducts which in this instance was located just at the liver margin. The distal end of the duct was found, but it was too small and fibrosed to use in an end to end repair. Accordingly a Roux Y arm of jejunum 24 inches in length was brought up posterior to the colon for direct anastomosis to the proximal duct, a vitallium tube inserted into each duct, and fixed in position with silk sutures; these tubes remained in position at least 18 weeks as shown by roentgenogram.

For eight months the patient was asymptomatic. Beginning January 1949 she began to complain of an occasional chill with fever and mild jaundice; however, the stools were brown. For the next 21 months attacks of pain began and became more severe and more frequent, but chills and fever were less prominent. Slight diarrhea was present during this interval, but no jaundice was present. About March 5, 1952 she had an attack of jaundice and acholic stools lasting for one day. Several days later the jaundice returned and persisted. However, the stools remained normal in color, thus making us think that perhaps one of the intrahepatic ducts was stenosed, whereas the other was open. Nevertheless we still thought intrahepatic disease most likely was the explanation of the jaundice. In view of persistent pain and jaundice we admitted her to the hospital in June 1952. At this time the prothrombin and cephalin flocculation tests were negative, but the thymol turbidity test was 29. The alkaline phosphatase test was 22.

The serum albumin was 4.4 and the globulin 3.8 Gm. per cent. The liver was enlarged 5 cm. below the costal margin and the spleen was palpable. Operation was done June 13,1952, but the stoma between the hilar ducts and arm of jejunum was wide open. Tissue for biopsy was taken from the liver. Microscopic examination revealed bile pigment in the small ducts and liver cells with some fibrosis, but no more than seen in any patient with obstructive jaundice. She was discharged June 25, 1952.

Because of persistent jaundice and severe pain she was admitted again to the hospital October 29. At this time the serum bilirubin was 15 and the icterus index 120. The prothrombin test was 77 per cent of normal. The alkaline phosphatase level was 17. Serum albumin

was 4.6 and globulin 4.4 Gm. per cent. Stools were occasionally pale, but usually of normal color; test for occult blood was positive. Pain persisted. The liver was palpable about 3 cm. below the costal margin.

In spite of the negative findings at operation June 13, we thought operation now advisable largely because of the prolonged jaundice and severe pain. At operation November 7, a definite stricture at the old stoma between the hilar ducts and arm of jejunum was found. This was forcibly dilated resulting in a sudden burst of arterial blood. We tied a small branch of the right hepatic artery but had to pack the area to control the bleeding completely. She was reoperated upon three days later for insertion of a prosthesis. The pack was removed and after gentle dilation a large polyethylene tube was inserted and sutured in place. Convalesence from this operation was satisfactory except for a wound dehiscence. She was discharged December 5, and for several weeks showed slight improvement. She then developed more pain and jaundice, but still had comparatively normal stools.

She was readmitted March 12, 1953 because of the increased pain and jaundice on the assumption that the stricture which was dilated Nov. 10, 1952 had recurred. The serum albumin was 3.5 and the globulin 2.2 Gm. per cent. The icterus index was 126. Prothrombin time was normal but the thymol turbidity test was 15. The alkaline phosphatase was 13. It was thought so much inflammation and scarring would be present at the liver hilus that a Longmire procedure now would be preferable. Accordingly, after intensive therapy with transfusions and forced feedings to correct the hypoproteinemia as much as possible, on March 23, 1953, a part of the left lobe of the liver was resected and an arm of jejunum brought up to anastomose with a dilated duct in the cut surface of the left lobe. Three days later, the patient vomited several hundred cc. of blood, and blood likewise escaped from the catheter in the left intrahepatic duct as well as from the wound alongside a drain. Prothrombin deficiency was considered as a cause of this bleeding but the prothrombin level was reported as 100 per cent. Numerous blood transfusions and intensive hykinone therapy were instituted and the bleeding ceased. The thymol turbidity varied from 11 to 23 and the cephalin flocculation test from 1 plus to 3 plus. On April 9 another severe hemorrhage from the wound developed and intense melena was noted. On April 13, the prothrombin time was 52 per cent of normal. Bleeding occured occasionally for the next several days until April 20 when it became more severe. Thinking that the cut surface of the liver might be the source of bleeding the wound was opened under anesthesia. Considerable bleeding was noted here and about the end of jejunum. These points were controlled and a pack inserted. From this date onward we had increasing difficulties controlling electrolyte deficiency, prothrombin deficiency and hypoproteinemia in spite of intensive therapy. The bleeding stopped following the operation April 20, but began again on April 23, on which date we opened the lower abdomen thinking perhaps the bleeding point might be an ulcer in the jejunum which was not exposable through the upper abdominal incision made at operation three days previously. However, no bleeding point and no ulcer was found. Her jaundice persisted and the hypoproteinemia increased in spite of numerous transfusions. She died May 10, 1953.

Autopsy revealed moderate subacute peritonitis and biliary cirrhosis with gastric erosions. The latter were considered to be terminal, and not the cause of the massive hemorrhages she had been having. She had a large thrombus attached to the wall of the left intrahepatic duct which was blocking the duct. Considerable purulent material was present in the duct proximal to the thrombus. It appears that the small catheter we had inserted into the left duct at the time we did the Longmire procedure had eroded the duct wall sufficiently to produce a tiny ulcer and hemorrhage.

Comment: During the last six months of this patient's life hepatic damage had been so severe as to produce jaundice with occasional acholic stools. Although we knew she had severe hepatic damage—because of positive liver function tests —we were unwilling to accept the information so completely as to eliminate the advisability of operation. Since the autopsy showed the opening between the

hilar ducts and the end of the jejunum was entirely open, it is obvious that our
dilatation and use of a prosthesis six months previously had been entirely suc-
cessful. The Longmire procedure was unnecessary. We should have paid more
attention to the stools which were cholic most of the time, but we thought one
intrahepatic duct was open and the other blocked by stricture. Actually this
was the case, as shown by the huge organized clot attached to the wall of the
duct and obstructing it. However, this clot probably developed a day or two
after our operation on March 23, 1953, and thus could not have been a part of
the clinical picture previous to this time.

Case 2. C. H., a white woman aged 62, entered The Illinois Research Hospital Feb. 2, 1948
with the history of having had a cholecystectomy elsewhere in May 1945 followed three
months later by pruritis and painless jaundice. Jaundice became intermittent, and pain in
the right upper quadrant soon was noted; chills and fever likewise developed. Stools were
acholic during the periods of jaundice. Since onset there was a weight loss of 40 pounds. In
August 1947, she had another operation elsewhere, at which time a narrowing of the com-
mon duct with a chronic pancreatitis was found. No relief was obtained from this opera-
tion; pain, intermittent jaundice, and an occasional chill persisted.

Upon admittance to our hospital she had jaundice, a moderate anemia, a serum albumin-
globulin ratio of 2.9/4.2 and a thymol turbidity of 20, indicating considerable hepatic
damage. The cephalin flocculation test was negative.

On February 6, we did a celiotomy. A fibrosed cord, representing the common duct, was
found, but no duct was demonstrable outside the liver. We aspirated the liver at the hilus
finding bile at a depth of 1½ inches. We excised liver tissue at this point attempting to ex-
pose and open the duct, but encountered (with our aspirating needle) the major trunk of
the right hepatic artery. Accordingly we discontinued our dissections hoping the several
points where we had aspirated the duct would open up and drain bile as they had on several
other occasions. Culture of the bile aspirated at the operating table yielded alpha prime hem-
olytic streptococcus and *E. coli*. Postoperatively, she did drain bile rather profusely, but
a persistent febrile reaction and a badly infected wound associated with rupture of three
intraabdominal abscesses, forced us to delay operation for several weeks.

After recovery from her septic complications from the operation in February 1948, she
was returned to the hospital. At this time she had slight jaundice, persistent fever, severe
anemia, tarry stools—due to hypoprothrombinemia—albumin–globulin reversal of 2.0/4.0
and a thymol turbidity test of 5.4. On July 23, 1948 another celiotomy was done. To our
surprise, at this time a dilated duct was found at the hilus; presumably the excavation we
had made in the liver at the previous operation had allowed a duct to project outward so
that it became readily visible. This stump was anastomosed to a Roux Y loop of jejunum.

A McArthur tube was left in as a prosthesis. Its passage was not noted by the patient.
Therefore we are not sure how long the tube remained in place—such tubes usually pass in
four to eight weeks. Following this procedure convalesence was surprisingly satisfactory.
By December 1948 she had gained 17 pounds in weight; had light brown stools and was no
longer having chills or fever. However, jaundice persisted, although slightly less in in-
tensity.

During the next several months numerous tarry stools were noted (due to hypopro-
thrombinemia). By September 1949, she had improved still more and the jaundice had
disappeared. When seen in May 1950, she gained a total of 38 pounds since operation;
stools had been brown persistently and no melena had been noted for months.

On Jan. 18, 1951, patient was readmitted to The Illinois Research Hospital with a history
of abdominal bloating of three months duration and melena of two days duration. The
prothrombin time was 52 per cent; serum albumin was 2.9 Gm. per cent and the serum
globulin 2.7 Gm. per cent. The non-protein nitrogen was 51. The liver edge was just pal-
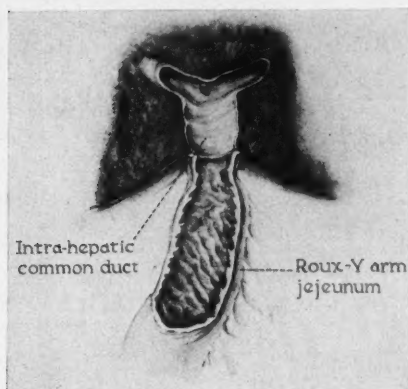
Fig. 5. Artist's drawing of the stoma (at autopsy) between the hilar duct and Roux Y arm of jejunum in case 2, revealing a wide open stoma. For two weeks before death the patient was jaundiced, had pale stools, and had bleeding from the nose as well as hematuria. The manifestations which resembled those seen in duct obstruction were obviously produced by the hepatic damage, which was of sufficient severity to cause death.

pable; the spleen could not be felt. During this observation of nine days there was no significant fever.

On Aug. 21, 1951 this patient again was readmitted with the history of being well until two weeks ago when she developed sharp mid abdominal pain radiating to the back, light stools, dark urine, and icterus. The temperature, pulse rate and respiratory rate were normal upon admission, but each rose precipitously until death on the third day of hospitalization. The history revealed a temperature of 100 to 103.5°F. with occasional chill during the past 10 days. The liver edge was obscured by distention, but was perhaps enlarged about 3 cm. below the costal margin. The thymol turbidity test was 24. Hematuria and bleeding from the nose and mouth were present during this admission. A blood culture revealed E. coli. The non-protein nitrogen was 65 and the CO_2 was 30 (acidosis). The albumin–globulin ratio was reversed. On the second day of hospitalization she became comatose and died the following day.

Autopsy revealed a suppurative cholangitis and two large multilocular abscesses (8 cm. in diameter) in the right lobe of the liver. The portal vein was partially occluded with a thrombus. Each lung was the seat of edema and bronchopneumonia. The anastomosis between the hilar duct and arm of jejunum was widely patent as illustrated in figure 5.

Comment: The cause of death in the patient described above was obviously the liver abscess and associated hepatitis although there was comparatively little biliary cirrhosis. However, it is a bit difficult to explain why the patient was comparatively free from symptoms for seven months prior to the final illness which lasted only two weeks. The liver abscess was so large it scarcely could have developed in such a short time as two weeks, unless a small abscess had been dormant for months and then became reactivated. The portal vein may have been the seat of a chronic infection which was responsible for the hepatitis and liver abscess. Previous to the interval of seven months, during which there was comparative freedom from symptoms, the patient had a period of illness lasting three months. During this period she was hospitalized, at which time she had a prothrombin time of 52 per cent and a slightly reversed albumin–globulin ratio,

indicating she had a very significant degree of hepatitis at that time. Presumably the damage done to the liver by the stricture, before its repair and correction slightly more than three years before death, had been irreparable. At least, we know she had evidence of severe liver damage for months before the successful repair of her stricture July 23, 1948, and no bile duct obstruction for three years before death.

SUMMARY

It is well known that most strictures of the common duct are secondary to operative trauma. Since they are common and not decreasing in frequency we must continue to emphasize the need for the utmost precautions during cholecystectomy. Important in these precautions are the need for an adequate incision, awareness of anomalies, safe mechanisms in controlling hemorrhage, and careful operating technic as has been discussed in detail.

Operations for strictures are extremely difficult and should be done only by surgeons fully experienced in surgery of the upper abdomen. Whenever possible, end to end anastomosis (after resection of the stricture) should be made, thus conserving function of the sphincter of Oddi. However, in the author's experience, construction of a Roux Y arm of jejunum with anastomosis of its end to the hilar duct yields results just as good as those following end to end anastomosis. In general, good to excellent results should be obtained in 60 per cent or more of patients. There is no agreement regarding the type of prosthesis to be used for support at the suture line. At present, the author is using no prosthesis when the duct is large, its wall comparatively thin and free from inflammation. When considerable inflammation is present in the duct wall, we resort to prolonged use of a prosthesis—8 to 10 months—particularly if the lumen of the duct is small. At present a T tube is used for this prosthesis even when supporting the anastomosis between the hilar duct and a Roux Y arm of jejunum, because it remains in position with much more certainty than a piece of rubber tube or a catheter.

Very important in the indication for operation for stricture of the common duct is the necessity of distinguishing duct obstruction from hepatic damage, as the cause of the jaundice and acholic stools. The hepatic damage usually is associated with a severe biliary cirrhosis and hepatomegaly, but not always. Hepatitis may produce jaundice without acholic stools, but as the disease progresses stools become acholic; if the lesion progresses to this stage prognosis for recovery is very poor. Extremely important in the differentiation of these two lesions are liver function tests. No single test is sufficiently accurate or sensitive to make this differentiation. In the author's opinion, the thymol turbidity test is much more sensitive and dependable than the others, but at least two others, such as the cephalin flocculation test and blood proteins—for level and reversal—are indicated. The prothrombin test often is very useful but subject to much inaccuracy especially when made by a relatively inexperienced technician.

Patients having only mild symptoms are not operated upon immediately

because many such patients have an occasional short attack of jaundice but recover completely without operative interference. The surgeon will be particularly reluctant to advise operation if the hilar duct used in the previous operation was short, small, and inflamed. Results in patients with ducts of this type are much less optimistic than when the duct is large, protruding beyond the hilus, and free from inflammation.

During the period of observation for mild symptoms liver function tests must be made at frequent intervals, and if they become positive, especially between attacks, operation then may be advisable. Occasionally, the portal hypertension created by the biliary cirrhosis is severe enough to cause hemorrhage from esophageal varices; if so, splenorenal or portacaval shunt may be indicated. Splenorenal shunt is usually preferable because the portal vein will be thrombosed so often in these patients.

REFERENCES

1. Cattell, R. B.: Benign strictures of biliary ducts, J. A. M. A. *134:* 235 (May 17) 1947.
2. Harridge, W. H.: Alterations in common bile duct stricture formation by proximal decompression, in Forum on Fundamental Surgical Problems, Clinical Congress of American College of Surgeons, 1952 session, pub. 1953, p. 411.
3. Lary, B. G., and Scheibe, J. R.: Effect of rubber tubing on healing of common duct anastomoses, Surgery *32:* 789 (Nov.) 1952.
4. Longmire, W. P., Jr., and Sanford, M. C.: Intrahepatic cholangiojejunostomy for biliary obstruction—further studies; report of four cases, Ann. Surg. *130:* 455 (Sept.) 1949.
5. Walters, W., and Kelly, A. H.: Surgical treatment of stricture of common and hepatic bile ducts; twenty-eight year survey, A. M. A. Arch. Surg. *66:* 417 (April) 1953.

GASTROJEJUNAL ULCER

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The problem of gastrojejunal ulcer always has been a serious one and the development of a jejunal or marginal ulcer has constituted the most serious complication in the surgical treatment of benign ulceration of the stomach and duodenum. The frequent development of gastrojejunal ulcer after surgical treatment of peptic ulcer is evidence of failure of surgical management and is, in general, the real reason for the development of the various surgical methods of treatment because our present methods have not proved completely satisfactory in all cases. As one reflects on the number of vagotomy operations that have been done in the past few years one wonders whether an increasing number of gastrojejunal ulcers resulting from gastro-enterostomy combined with transabdominal vagotomy will not be encountered, since gastrojejunal ulcer has developed much more frequently after gastro-enterostomy than after a high gastric resection. Too little time has elapsed since the use of this method of surgical treatment since, in our experience, ulcer has recurred as long as 32 years after the original operation. At the Lahey Clinic over a period of 14 years we have operated upon 258 patients with symptoms arising from either malfunctioning gastro-enterostomy or jejunal ulcer proved by operation. In this group of 258 cases there were 172 cases of definite jejunal ulcer. We are limiting our discussion to this group in which jejunal ulcers were definitely demonstrated to be present at operation. In the majority of patients the jejunal ulcer occurred after gastro-enterostomy; in 133 patients (77.3 per cent) the ulcer arose after gastrojejunostomy and in 39 patients (22.7 per cent) it followed gastrectomy. Of this group of 172 cases of proved gastrojejunal ulcer after either gastro-enterostomy or gastric resection, 14 patients, or 8.1 per cent, had previously been operated upon at the Lahey Clinic. During this same period of 14 years there had been a total of 1203 resections for peptic ulcer, duodenal, gastric or jejunal, and 135 gastro-enterostomies. There were 14 proved jejunal ulcers in a group of 1166 resections or gastro-enterostomies (omitting the 172 gastrojejunal ulcers), an incidence of about 1 per cent. Unquestionably, this figure would be much larger if all of those cases of either resection or gastro-enterostomy could be carefully followed roentgenologically and by study of postoperative symptoms over the years, and if the unoperated cases could be added—those in which the diagnosis was based on roentgen examination—so that such a low figure as 1 per cent for recurrent ulcer in our group is entirely too low. Certainly, from our experience, and according to statistics of many published

reports, the incidence is much higher following gastro-enterostomy than after an adequate gastric resection.

Priestley and Gibson,[13] in a recent paper, stated that the average incidence of recurrent jejunal ulcer after gastro-enterostomy for duodenal ulcer, as reported in the literature, is approximately 15 per cent. The reported incidence has varied from 2.8 per cent as given by Judd[8] and Hoerner up to 34 per cent as reported by Lewisohn[11] in a careful study in 1925. Wilkie[18] reported an incidence of 3.5 per cent following gastro-enterostomy and Wangensteen[17] and Lahey[10] gave the incidence after gastro-enterostomy as approximately 15 per cent.

Gastrojejunal ulcer may develop after gastro-enterostomy, gastric resection or any operative procedure in which the jejunum is anastomosed to the stomach. The frequency with which jejunal ulcer has developed after partial gastrectomy has varied in the many reports in the literature, but the average incidence is considerably lower than following gastro-enterostomy. Kiefer,[9] in 1942, reported an incidence as high as 6.8 per cent from this clinic, 3.4 per cent of which were proved by operation and an equal percentage was diagnosed by roentgenologic examination. Reinhoff,[16] in a series of 266 patients followed 2 to 10 years, reported that 11.1 per cent required secondary surgical treatment chiefly because of jejunal ulceration.

Gastrojejunal ulcers most commonly arise after surgical treatment of duodenal ulcer. In this series of 172 cases (table 1), the original lesion was duodenal ulcer in 154 (89.5 per cent), gastric ulcer in 5 (2.9 per cent) and the location of the ulcer was not recorded in 13 (7.6 per cent). Priestley[13] and Gibson stated that 92.5 per cent occurred after operation for duodenal ulcer, 1.4 per cent after gastric ulcer and 5.3 per cent after peptic ulcer, type unknown.

ETIOLOGY

No satisfactory explanation for the development of gastrojejunal ulcer has been proposed, but the cause of jejunal ulcer probably is similar to that of duodenal ulcer. It is well known that the tendency toward damage of the mucosa by acid increases as the distance from the pylorus is increased, as has been shown by the investigations of Mann[12] and Bollman. Such increased susceptibility of the jejunal mucosa to injury by hyperacidity and hypersecretion in a young person predisposes to formation of an ulcer following any surgical procedure in which the jejunum is anastomosed to the stomach. On the other hand, it is of interest to observe that ileal ulcer infrequently occurs with accidental gastro-ileostomy. We have operated on 7 patients who had faulty anastomoses of the ileum to the

TABLE 1

172 gastrojejunal ulcers

Type of Original Ulcer	Number	Per cent
Duodenal ulcer	154	89.5
Gastric ulcer	5	2.9
Type not stated	13	7.6

stomach when gastrojejunostomy was intended and in none of these patients was an ulcer present at the gastro-ileal stoma.

This tendency to the development of ulcer perhaps is increased by carelessness in diet following surgical treatment of the ulcer and also by the indiscriminate use of alcohol and tobacco; emotional stress and the tendency to abandon all restriction in diet and irregular habits of living undoubtedly play a part in the development of recurrent ulcer. It is extremely important to emphasize to the patient who is to undergo gastric resection that great attention postoperatively must be paid to diet and to habits of living in order to avoid recurrence of an ulcer. It is our opinion that such directions are not sufficiently stressed to the patient who ultimately must come to surgery for relief of the complications of chronic peptic ulcer or the pain of an intractable ulcer.

Many factors influence the development or aid in the prevention of a jejunal ulcer after surgical treatment. It is well recognized that the majority of jejunal ulcers occur after gastro-enterostomy (fig. 1). In this group of 172 cases, 77.3 per cent occurred after gastrojejunostomy, and the high occurrence rate after gastro-enterostomy already has been referred to previously. Another factor is the location of the original lesion. In this group of 172 cases the original operation was done for duodenal ulcer in 154 patients (89.5 per cent), gastric ulcer in 5 patients (2.9 per cent) and in 13 patients (7.6 per cent) the site of the

FIG. 1   FIG. 2

FIG. 1. Gastrojejunal ulcer in a patient aged 57. Gastro-enterostomy was done three years before admission to clinic. Large jejunal ulcer was found 6 or 7 cm. distal to the stoma in the jejunal loop. Ulcer had perforated into mesentery of jejunum. Resection was done. (Courtesy of Postgraduate Medicine, *12:* 139, August, 1952.)

FIG. 2. Gastrojejunal ulcer after inadequate gastric resection five months before admission. Only the antrum was removed; a large gastric stump remains. Resection was done. A jejunal ulcer was found 4 cm. distal to the stoma in the distal jejunal loop; the ulcer had perforated into the transverse colon. (Courtesy of W. B. Saunders Co., Surgical Clinics of North America, *26:* 753, June, 1946.)

peptic ulcer was not stated. Again, a sufficiently radical resection should be done in order to obtain a relative achlorhydria, and at least 70 per cent of the stomach should be resected. Most of the jejunal ulcers that occurred after partial gastric resection in this series were found in patients in whom a highly insufficient resection of the stomach was done (figs. 2 and 3).

We have had a high incidence of recurrent ulcer after an exclusion type of operation, such as the Finsterer method of exclusion. In our experience 54 per cent of the exclusion operations have resulted in gastrojejunal ulcers. Ransom[14] has stated that the exclusion operation has been a most disappointing procedure and that jejunal ulcers occurred in 30 to 40 per cent of those cases in which

FIG. 3. Gastrojejunal ulcer after inadequate resection five years before admission. Note that two-thirds of the proximal portion of the stomach remains. Jejunal ulcer was found 3 cm distal to the stoma, and had perforated into the mesentery. Resection was done.

such an operation had been used. The use of a short proximal jejunal loop probably is of some importance in the development of jejunal ulcer, but perhaps has been overemphasized. The jejunal loop forming the anastomosis should be made as long as is necessary to prevent tension resulting from attachment of the jejunum in a high resection of the stomach, but should be made as short as possible in order not to eliminate too much of the jejunum for small bowel absorption. It has been our experience that an anastomosis anterior to the colon can be made with a very short loop and is not an item of great significance in the prevention of the development of a gastrojejunal ulcer. This particularly is true if the gastrocolic omentum is removed from the colon and resected with the stomach, as we do in all cases in which the omentum is thick and fatty.

We also believe that the removal of a duodenal ulcer is an important part of a gastric resection, and in most instances this can be done with safety. In those cases in which the duodenal ulcer is low or in the second part of the duodenum, the ulcer can be removed with few exceptions and a safe closure of the duodenal stump can be made without impingement upon the ampulla of Vater. The position of the ampulla of Vater readily can be determined by exposing the common bile duct, inserting a rubber catheter (fig. 4) into the duct and threading it down through the ampulla of Vater into the duodenum so that injury of the common bile duct or ampulla of Vater cannot result with the dissection. With this catheter in place, the common bile duct or the ampulla of Vater easily can be identified.
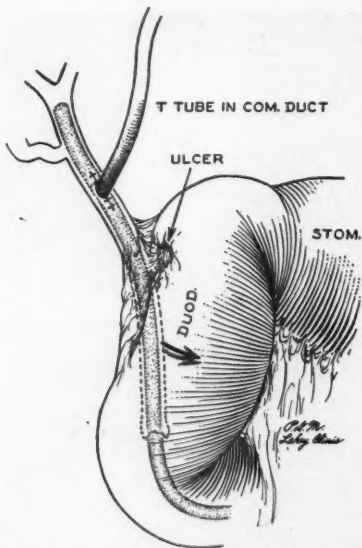
FIG. 4. Common bile duct is exposed, opened, and catheter inserted into the duct and passed down into the duodenum. This procedure aids in excision of ulcers low in the duodenum with considerable induration about the duct or ampulla of Vater. (Courtesy of W. B. Saunders Co., Surgical Clinics of North America, *32*: 822, June, 1952.)

The exclusion operation is never used unless a transection of the duodenum can be made above the ulcer in the duodenum—not above the pyloric ring which allows antral mucosa to remain—and this exclusion of the ulcer is advisable only when there is no obstruction in the duodenum at the level of the ulcer, which is allowed to remain because of the technical difficulties associated with excision.

AGE AND SEX

Jejunal ulcer may occur at any age. The youngest in our group was 22 years and the oldest was 75 years; the average age was 49 years. Ransom stated that the average age in his group of 81 patients was 48.4 years.

In regard to sex, the largest percentage of patients is males. In our series

TABLE 2

Symptoms

	Number	Per cent
Pain	138	80.0
Nausea and vomiting	61	35.0
Diarrhea	29	17.0
Weight loss	65	38.0

of 172 patients, 157 or 91.3 per cent were males, and 15 or 8.7 per cent were females. Ransom noted that 85.2 were males and 14.8 per cent were females in his series. Glenn,[6] in a series of 113 patients, reported 88.5 per cent males and 11.5 per cent females. This ratio corresponds very closely to the sex incidence of chronic peptic ulcer other than gastrojejunal ulcer.

Symptomatology

The symptoms of a jejunal ulcer are more or less identical with the symptoms of the original ulcer. In this series of 172 patients the symptoms consisted of pain in 138 or 80 per cent, nausea and vomiting in 61 patients or 35 per cent, diarrhea in 29 patients or 17 per cent and weight loss in 65 patients or 38 per cent (table 2). Hemorrhage also occurred in 43 per cent of these patients.

The time interval between the original operation and the onset of symptoms varied from 2 weeks up to 32 years; the average length of time was 6 years. The time interval between the original operation and the operation for jejunal ulcer averaged $9\frac{1}{2}$ years; the shortest was 6 months and the longest 33 years. Allen[1] and Welch stated that one-third of their cases of jejunal ulcer occurred in the first year after operation, one-third in the second year and the remainder at a later period. The average time after gastro-enterostomy in Priestley and Gibson's series was 3.7 years and after resection was 1.7 years. In 56 per cent of their patients the ulcer developed within the first two years. There is a considerable tendency to greater severity of symptoms and to a more rapid progressiveness of the clinical course. In our group of cases pain has been the most consistent symptom and is usually more severe and intractable than that caused by the original ulcer. The pain is usually to the left of the midline and food and antacids do not give relief to the same extent as with the original ulcer.

The diagnosis can be established on the basis of the symptoms and upon the roentgenologic findings, but it is important to emphasize the fact that a gastrojejunal ulcer may occur after any type of anastomosis in which the jejunum is attached to the stomach, whether after resection or after gastro-enterostomy. The examining physician always should be suspicious of the possibility of the development of a jejunal ulcer in a patient who gives a history of a previous gastric operation and in whom pain develops to the left of the midline and at about the level of the umbilicus. Hemorrhage is an extremely important symptom; is usually indicative of recurrent ulcer or an acute mucosal ulceration and indeed may be the first symptom of a gastrojejunal ulcer.

ROENTGENOLOGIC DIAGNOSIS

The roentgenologic diagnosis of a gastrojejunal ulceration presents many more difficulties than that of the original chronic peptic ulcer. This is attributable to several factors: the difficulty in clearly demonstrating an ulcer niche and the tendency for superimposed coils of small intestine to prevent visualization of the ulcer in the gastrojejunal stoma. Feldman[5] pointed out that the reliability of the diagnosis depends upon a very careful study of the gastrojejunal area and proposed a number of characteristic signs that are suggestive of gastrojejunal ulcer. The reader is referred to this article on roentgen diagnosis.

Eusterman, Kirklin and Morlock[4] pointed out that the roentgenologic demonstration of a nonfunctioning gastroenteric stoma is a significant phenomenon and found in the study of 62 cases that in 13 per cent nonfunction was due to obstruction and in 77 per cent a gastrojejunal ulcer or gastrojejunitis was present. Priestley and Gibson, in 1948, reported a definite roentgen diagnosis in only 52 per cent of 270 cases but that roentgenographic criteria suggestive of ulcer were present in 86 per cent of the 270 cases proved by operation. In our series of 172 cases the roentgenologic evidence was positive in 117 cases, or 72 per cent, and negative in 46 cases, or 28 per cent, with all these jejunal ulcers being verified later by operation.

COMPLICATIONS

The complications of a gastrojejunal ulcer are hemorrhage, perforation, obstruction and the development of a gastrocolic fistula (table 3).

One of the most frequent and serious complications is hemorrhage, and, in any patient who has had a gastric operation and who has a hemorrhage, the likelihood of the presence of a jejunal ulcer is considerable. Hemorrhage is more frequent with jejunal ulcer than with chronic duodenal or gastric ulcer. Wilkinson and Tracey,[19] in this clinic, however, reported that in a study of 140 patients with peptic ulcer, the first symptom was gross hemorrhage in 70. Eusterman reported gross hemorrhage in 17 per cent of patients operated upon at the Mayo Clinic for duodenal ulcer. Eliason and Ebeling[3] found that acute hemorrhage occurred in 19.5 per cent of 546 cases of duodenal ulcer. Gordon-Taylor[7] pointed out that hemorrhage from an anastomotic or jejunal ulcer is much more frequent than from a duodenal ulcer, and that in 50 per cent of the cases there is a tendency to bleed, but the mortality rate is low. He counseled conservative measures unless hemorrhage is very severe and appears to be

TABLE 3

Complications of jejunal ulcer

	Number	Per cent
Hemorrhage	74	43.0
Perforation	19	11.0
Obstruction	9	5.2
Gastrocolic fistula	32	18.6

FIG. 5   FIG. 6

FIG. 5. Gastrojejunocolic fistula in a patient aged 51. Gastro-enterostomy had been done 13 years before admission. Roentgenogram after a barium meal shows that the barium passed into the jejunum and colon. Two stage resection was done.

FIG. 6. Gastrojejunal ulcer with impending fistula into the colon in a patient aged 58. Gastro-enterostomy was done five years before admission to the clinic. Note crater in inferior margin of stoma. At operation, considerable induration of the wall of the transverse colon was found but no penetration; ulcer was adherent to colon. (Courtesy of Postgraduate Medicine, *12:* 141, August, 1952.)

uncontrollable. Our incidence of hemorrhage of 43 per cent in this group of 172 cases of jejunal ulcer very closely parallels Gordon-Taylor's experience.

Perforation occurred in 19 patients, or 11 per cent of the total group, and is a frequent complication of jejunal ulcer (figs. 1 and 2). Free perforation into the peritoneal cavity rarely occurs, and we have had no such case. Ordinarily, perforation consists of penetration of the adjacent organs—onto the colon, onto the mesocolon, into the abdominal parietes, the pancreas and the liver. It is important to remember that with the development of the jejunal ulcer and with involvement and penetration of the colon, the development of a gastrocolic fistula is highly probable and is a serious complication of such a jejunal ulcer (fig. 5). Fistula has occurred in 32 of the series of 172 cases, or in 18.6 per cent. Should evidence of colon irritability develop in a patient who has had a gastric operation, the possibility of penetration onto the wall of the colon with subsequent development of a gastrocolic fistula should be seriously considered and an operation should be carried out promptly (fig. 6).

Obstruction is a common occurrence and although it was found in only 9 patients (5.2 per cent), there is a larger group (86 cases) of malfunctioning gastro-enterostomy stomas which we have not included in this series. In this group of 86 patients the malfunctioning stoma was in a large measure the result of stenosis or obstruction and was indicative of a previous jejunal ulcer which healed, with resulting cicatrization of the stomal orifice. With obstruction of the gastro-enterostomy stoma, reactivation of the original ulcer frequently

has occurred, and in many cases the symptoms are attributable to an active duodenal ulcer and not to the gastro-enterostomy.

TREATMENT

Gastrojejunal ulcer, similar to gastric or duodenal ulcer, may respond to medical treatment and under a careful regimen in the hospital may heal quite readily. However, many jejunal ulcers are intractable to conservative medical therapy and these patients early come to surgery if the ulcer fails to show evidence of healing. It must be noted, however, that many of the lesions at the gastrojejunal stoma, particularly those arising after gastric resection, are mucosal erosions or acute superficial ulcers, some of which will respond quite satisfactorily to medical measures. Medical treatment should be carried out in the hospital and if healing fails to occur, as evidenced by alleviation of symptoms or by disappearance of the ulcer niche, surgical treatment should be instituted early. Priestley stated that if the diagnosis of a jejunal ulcer has been established, surgical intervention is advised and indicated at once. It is our opinion that many patients with early jejunal ulcers resulting after partial gastrectomy should be given a reasonable period of medical treatment, but it is evident that prolonged medical treatment is not completely satisfactory, and it is well recognized that the jejunal ulcer may fail to respond to medical treatment. The physician always must keep in mind that serious complications may result if surgical intervention is not carried out when the ulcer fails to respond to ordinary

Fig. 7. Gastrojejunal ulcer. Two previous resections had been done; gastric antrum was not removed; only a small remnant of stomach remains.
  a, Preoperative roentgenogram; arrow indicates jejunal ulcer.
  b, Forty-eight days later, after removal of gastric antrum. No other definitive surgical procedure done. Ulcer healed and jejunal peristalsis is normal. (Courtesy of W. B. Saunders Co., Surgical Clinics of North America, 26: 761, June, 1946.)

Fig. 8. Jejunal ulcer following resection five years previously. Note that 70 per cent of the stomach is resected. The ulcer is situated in the distal loop of the jejunum.

medical measures. The tendency to use conservative medical measures after repeated recurrences of jejunal ulcer is to be condemned.

It is our impression that partial gastrectomy is the standard procedure for gastrojejunal ulcer arising after a gastro-enterostomy, and will result in the highest percentage of cures. When an ulcer recurs after a gastrectomy, frequently it is true that resection was not sufficiently radical. This can be demonstrated by roentgenogram (figs. 3 and 4), and if the resection is inadequate a further resection should be done. At the time of another resection, however, the operating surgeon should look for an unremoved antrum (fig. 7), which may account for the recurrence of an ulcer when the first resection was adequate. Such a recurrent ulcer has been noted in 8 of our patients in whom the antrum had not been removed; in 3 patients the remaining part of the stomach was not resected and the jejunal ulcer healed after removal of the gastric antrum alone (fig. 8). When resection has been adequate and another ulcer has developed, we believe that a vagotomy should be done, preferably through the abdomen, at which time the surgeon can search for an unremoved antrum. We believe that the use of vagotomy is of value only if the antrum has been removed and if a radical resection of the stomach has been done.

The Report of the Committee on Surgical Procedures of the American Gastroenterological Association[15] concluded that vagotomy has value in the treatment of patients with gastrojejunal ulcer following gastric resection, but in those ulcers following gastro-enterostomy the results of resection alone were better. We again would agree with this opinion if a high radical resection, or a re-resection, had been done prior to the development of a jejunal ulcer, and we believe such a vagotomy is an absolute necessity in an effort to prevent the future development of recurrent ulcer. Crile[2] reported that abdominal vagotomy with or without revision of the stomal orifice has given good results in 23 of 27 patients. There were presumably 4 recurrences in 27 patients, an incidence of 15 per cent, which is a high rate.

TABLE 4

Operations for jejunal ulcer—172 patients

Partial gastrectomy and resection of jejunum	62
Partial gastrectomy and closure of jejunal stoma	27
Re-resection of stomach and resection of jejunum	16
Partial gastrectomy, resection of jejunum, vagotomy	6
Re-resection and resection of jejunum and vagotomy	6
Removal of pyloric antrum (3 with other procedures)	8
Vagotomy (21 with other procedures)	25
Jejunal ulcer	148
Gastrojejunocolic fistula (stage operation)	55
Total operations	203

TABLE 5

Postoperative deaths

Deaths after operation for jejunal ulcer		4
Sepsis	2	
Coronary accident	1	
Hemorrhage (4 weeks postoperatively)	1	
Deaths after second stage for gastrojejunocolic fistula		2
Total		6 (2.9%)

In this group of 172 cases of gastrojejunal ulcer there has been a total of 203 operations; 148 operations for jejunal ulcer and 55 operations in stages for gastrojejunocolic fistula. Sixty-two patients had partial gastrectomy and jejunal resection; 27 had partial gastrectomy and closure of the jejunum; 16 had re-resection and jejunal resection; 6 had partial gastrectomy and jejunal resection plus vagotomy; and 6 had re-resection and jejunal resection plus vagotomy. Eight patients had resection, with the antrum and ulcer unremoved. Vagotomy was done in 25 patients, in 21 of whom the operation was combined with other procedures (table 4).

The procedure mortality in this group of 172 cases with 203 surgical procedures was 2.9 per cent (6 postoperative deaths). Four deaths followed operations for jejunal ulcer; 2 followed a second stage operation for gastrojejunocolic fistula, one of which resulted from peritonitis three months after the second stage (table 5). One patient who had had two resections, plus a transthoracic vagotomy, ultimately had to have a total gastrectomy to prevent the recurrence of ulcer; this patient now has been well for a period of five years. Total gastrectomy was done in a second patient because of the occurrence of ulcer after two previous resections of the stomach.

We have been able to follow 149 of these patients; in 23 there has been no follow-up except by letters, which indicate no recurrence, and the remainder of the patients have been followed very carefully by roentgenologic and clinical examinations. Jejunal ulcer has recurred in 10 patients; 1 of these 10 patients had had a vagotomy plus resection. There has been one recurrence after a gastrojejunocolic fistula and one recurrence following vagotomy combined with a conservative gastric operation.

SUMMARY AND CONCLUSIONS

In summary, a study of 172 cases of gastrojejunal ulcer has been presented. In only 14 patients was the original operation done at the Lahey Clinic. The time interval between the original operation and onset of symptoms varied from two weeks to six years.

High partial gastrectomy has been the standard procedure for the treatment of gastrojejunal ulcer occurring after gastro-enterostomy. Re-resection has been used in jejunal ulcer occurring after resection of the stomach if the previous resection was inadequate.

When an adequate resection previously has been carried out, an intra-abdominal vagotomy is done and search is made for an unremoved antrum. We have found it necessary to do total gastrectomy in 2 patients in whom ulcer recurred after extremely radical resections of the stomach and vagotomy.

The operative mortality was 2.9 per cent (6 cases in 203 surgical procedures).

REFERENCES

1. Allen, A. W., and Welch, C. E.: Gastric resection for duodenal ulcer; follow-up studies' Ann. Surg. *115:* 530 (April) 1942.
2. Crile, G., Jr., and Brown, G. M., Jr.: Vagotomy as treatment for marginal ulcer, Gastroenterology *17:* 14 (Jan.) 1951.
3. Eliason, E. L., and Ebeling, W. W.: Catastrophes of peptic ulcer, Am. J. Surg. *24:* 63 (April) 1934.
4. Eusterman, G. B.; Kirklin, B. R., and Morlock, C. G.: Nonfunctioning gastroenteric stoma; diagnostic study of sixty-two surgically demonstrated cases, Am. J. Digest. Dis. *9:* 313 (Oct.) 1942.
5. Feldman, M.: Clinical Roentgenology of the Digestive Tract, ed. 3, Baltimore, Williams & Wilkins Company, 1948.
6. Glenn, F.: Secondary operations for peptic ulcer, Ann. Surg. *132:* 702 (Oct.) 1950.
7. Gordon-Taylor, G.: Present position of surgery in treatment of bleeding peptic ulcer (Lettsomian lecture), Brit. J. Surg. *33:* 336 (April) 1946.
8. Judd, E. S., and Hoerner, M. T.: Jejunal ulcer, Ann. Surg. *102:* 1003 (Dec.) 1935.
9. Kiefer, E. D.: Jejunal ulcers and recurrent hemorrhages after partial and subtotal gastrectomy for peptic ulcer, J. A. M. A. *120:* 819 (Nov. 14) 1942.
10. Lahey, F. H.: Inflammatory lesions of stomach and duodenum, J. A. M. A. *127:* 1030 (April 21) 1945.
11. Lewisohn, R.: Frequency of gastrojejunal ulcers, Surg., Gynec. & Obst. *40:* 70 (Jan.) 1925.
12. Mann, F. C., and Bollman, J. L.: Experimentally produced peptic ulcers: development and treatment, J. A. M. A. *99:* 1576 (Nov. 5) 1932.
13. Priestley, J. T., and Gibson, R. H.: Gastrojejunal ulcer; clinical features and late results, Arch. Surg. *56:* 625 (May) 1948.
14. Ransom, H. K.: Treatment of jejunal ulcer; comparative follow-up study, Arch. Surg. *58:* 684 (May) 1949.
15. Report of the Committee on Surgical Procedures of the National Committee on Peptic Ulcer, American Gastroenterological Association, Gastroenterology *22:* 297 (Nov.) 1952.
16. Rienhoff, W. F., Jr.: Analysis of results of surgical treatment of two hundred and sixty consecutive cases of chronic peptic ulcer of duodenum, Ann. Surg. *121:* 583 (May) 1945.
17. Wangensteen, O. H.: Role of surgeon in management of peptic ulcer, New England J. Med. *236:* 191 (Feb. 6) 1947.
18. Wilkie, D. P. D.: Jejunal ulcer: some observations on its complications and their treatment, Ann. Surg. *99:* 401 (March) 1934.
19. Wilkinson, S. A., and Tracey, M. L.: History of hemorrhage in peptic ulcer, Gastroenterology *7:* 450 (Oct.) 1946.

MEDIASTINAL TUMORS

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Chest roentgenograms of a large portion of the population are being made in an effort to detect neoplasms, tuberculosis, and other forms of pulmonary infection. As a result of these yearly surveys and the more frequent use of chest roentgenograms in evaluating patients clinically, an increasing number of mediastinal tumors are being detected. Many of these tumors are malignant or may produce serious complications if untreated. The purpose of this discussion is to review some clinical observations regarding mediastinal tumors which may be useful in their early diagnosis and proper management.

From 1946 through 1952, 97 patients with mediastinal tumors, whose diagnoses were established surgically, were treated at Scott and White Clinic. Table 1 shows those mediastinal tumors treated with irradiation. Table 2 lists those which were excised. There was one death in the surgically treated group.

Numerous classifications of mediastinal tumors have been proposed, depending upon cytology, embryology, location in the mediastinum, and whether congenital or acquired. For simplicity, it is convenient to classify them into either anterior, superior, or posterior mediastinal tumors according to their usual location in the mediastinum.

ANTERIOR MEDIASTINAL TUMORS
Lymphoblastomas

These are the most common tumors in the anterior mediastinum and include lymphosarcoma and Hodgkin's disease.

Lymphosarcoma. Intrathoracic lymphosarcoma may originate in mediastinal lymph nodes or in the thymus. As the lesion progresses, the superior mediastinum may be filled—with eventual extension into the neck. In the advanced case, symptoms may be due to compression of great vessels, the esophagus, bronchi, or the trachea.

The roentgenologic appearance of lymphosarcoma is fairly characteristic. In addition to the anterior mediastinal lymph nodes, the perihilar nodes—unilaterally or bilaterally—often are involved, resulting in a slightly nodular mass in the anterior mediastinum. When the involvement is bilateral, lymphosarcoma is even more probable.

Hodgkin's disease. This disease seldom is confined to the mediastinum and usually is accompanied by cervical or axillary lymphadenopathy. In all of the

MEDIASTINAL TUMORS TREATED WITH IRRADIATION

No. of Patients

Lymphosarcoma 36

Hodgkin's disease 8

Metastatic malignancy 27

MEDIASTINAL TUMORS TREATED SURGICALLY

No. of Patients

Neurogenic tumor................. 7
Lipoma........................ 5
Teratoid tumor 2
Thymoma 2
Hyperplasia of thymus 1
Carcinoma, type undetermined 1
Esophagenic cyst 1
Bronchogenic cyst 1
Thyroid (fetal and colloid adenoma)..... 1
Hodgkin's disease................. 1
Mesothelioma 1
Glomus tumor 1
Chondroma 1
Tuberculoma................... 1

lymphomas, the diagnosis often may be established by microscopic examination of an excised lymph node. When there are no involved lymph nodes available, the process is bilateral, and the diagnosis of lymphoblastoma seems very probable, one may use a diagnostic and therapeutic trial of roentgenotherapy. If there is not definite reduction in the size of the lesion in one month, exploratory thoractomy is indicated. Once the diagnosis of lymphosarcoma or Hodgkin's disease has been established, roentgenotherapy usually is the treatment of choice. For refractory cases, nitrogen mustard has proved useful (fig. 1).

Teratoid Tumors

Harrington[3] conveniently classified both dermoid tumors and teratomas as teratoid tumors since he thought most of them actually had some derivation from all three germinal layers. These tumors, which may be cystic or solid, are slow growing and seldom are seen before puberty. They usually originate in the midline and in the upper anterior mediastinum, but become displaced to one side as they increase in size. Occasionally, the larger lesions appear to extend into each side of the thorax. These tumors may compress the trachea and displace it posteriorly. Their sharp demarcation, round or oval shape, and position in the

anterior mediastinum all suggest proper identity. Detection of calcium or bone in a cyst is of great diagnostic value.

Symptoms usually are due to pressure on surrounding structures; but, occasionally, a cyst will rupture into a bronchus with expectoration of hair, sebaceous material, and other contents of the cyst. Infection within the cyst or pleural emphyema subsequent to rupture of the cyst are frequent complications.
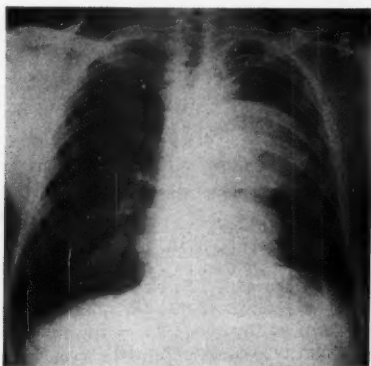
FIG. 1. Large unilateral anterior mediastinal tumor found at thoracotomy to be Hodgkin's disease.

The incidence of malignancy in teratoid tumors has been reported in various series as being from 10 to 40 per cent.[2, 6] Rusby[6] pointed out that if these tumors are not removed, they almost always contribute to the death of the patient. Heuer and Andrus[4] found that 47 of their 48 patients with untreated teratoid tumor died because of the tumor. These neoplasms should be completely excised as soon as their presence is detected. Irradiation has not proved useful except
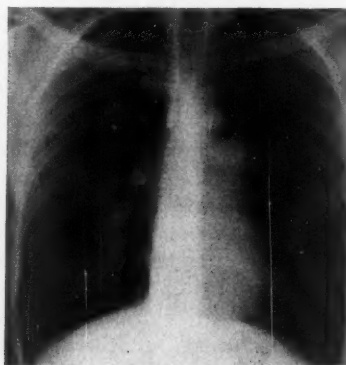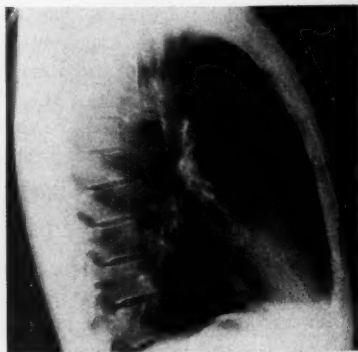
FIG. 2   FIG. 3
FIG. 2. Teratoid tumor in anterior mediastinum
FIG. 3. Lateral chest roentgenogram (same patient as in fig. 2) demonstrating tumor in the upper anterior mediastinum. Visible calcification in the wall of the tumor suggests the proper diagnosis.

for temporary palliation in a few patients with inoperable malignant tumors (figs. 2 and 3).

Tumors of the Thymus

Benign hyperplasia of the thymus, although not a true neoplasm, may cause dyspnea, cyanosis, cough, and other respiratory symptoms in the infant. Roentgenographic diagnosis, at times, is difficult, as the normal thymus may appear fairly large. If lateral and oblique views reveal tracheal compression and the patient has compatible symptoms, a trial of irradiation therapy is permissible. Occasionally, thymectomy may be necessary if symptoms persist.

The thymus may be the source of benign and malignant neoplasms. The majority of the benign tumors are associated with myasthenia gravis. Malignant thymomas more often produce pressure symptoms such as cough, dyspnea, and
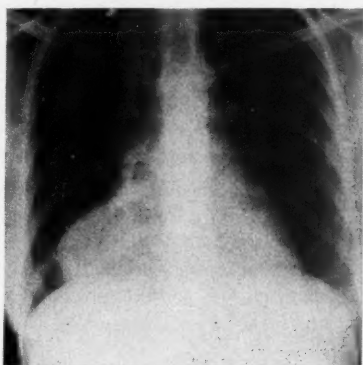
FIG. 4. Thymoma of the mediastinum in an uncommon position. More often, these tumors are in the upper anterior mediastinum.

pain. Seybold,[8] in reporting the experience of the Mayo Clinic, found that 15 per cent of their patients with myasthenia gravis had thymic neoplasms and 75 per cent of thymic neoplasms occurred in patients with myasthenia. These tumors nearly always are located in the anterior or superior mediastinum, but have been reported in the posterior and inferior mediastinum (fig. 4). Deposits of calcium are fairly common, but certainly are not diagnostic. Roentgenograms usually will reveal the presence of thymic neoplasms. Surgical extirpation is indicated if the patient is a suitable risk and evidence of metastasis is absent.

Pericardial Celomic Cysts

Pericardial celomic cysts of Lambert probably are due to faulty development of the pericardium and consist of thin-walled cysts filled with clear fluid. They may be visualized anywhere along the pericardium, but usually are found in the cardiophrenic angle, slightly more frequently on the right. These lesions generally are benign, but should be removed because of the possibility of malignancy in any undiagnosed mediastinal neoplasm.
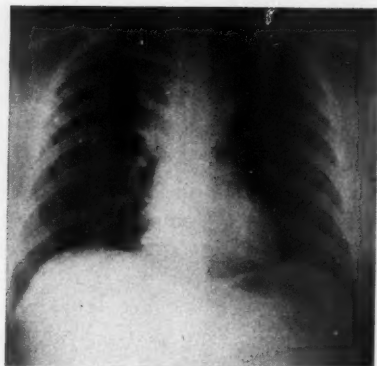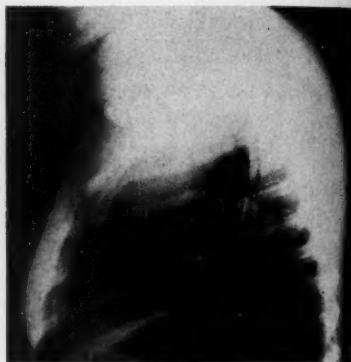
Fig. 5   Fig. 6
Fig. 5. Benign nontoxic nodular thyroid tumor in the anterosuperior mediastinum
Fig. 6. Lateral view (same patient as figure 5) demonstrating a well circumscribed neoplasm in the anterosuperior mediastinum.

SUPERIOR MEDIASTINAL TUMORS
Thyroid Tumors

Substernal extension of a thyroid tumor is not rare, but a completely mediastinal location of this lesion is uncommon. Most intrathoracic thyroid tumors lie in the anterosuperior mediastinum, but may occur in the posterosuperior mediastinum. Occasionally, motion of the tumor on swallowing may be detected fluoroscopically, suggesting the diagnosis. Radioactive iodine also may be useful in detecting these lesions especially if hyperthyroidism is present. Symptoms may be due to hyperthyroidism or to compression of the esophagus or trachea (figs. 5 and 6).

Mediastinal goiters should be excised either for relief of associated symptoms or because of the possibility of malignancy.

Bronchogenic Cysts

Bronchogenic cysts may occur in any portion of the mediastinum, but most often are in the posterosuperior mediastinum near the bifurcation of the trachea. These tumors are round or ovoid in shape, and may resemble a teratoid tumor or a neurogenic neoplasm. A patent communication with the tracheobronchial system seldom can be demonstrated, but bronchography and tomography may be useful in establishing the position and nature of the tumor.

Bronchogenic cysts usually are asymptomatic, but, occasionally, may become infected or produce symptoms from pressure on surrounding structures. Because of the possibilities of malignancy, infection, or pressure symptoms, these neoplasms should be removed.

POSTERIOR MEDIASTINAL TUMORS
Neurogenic Tumors

The most common primary tumors of the posterior mediastinum, excluding carcinoma of the esophagus, are those of neurogenic origin. Many classifications have been given, and the one used here was proposed by Sabiston and Scott.[7]
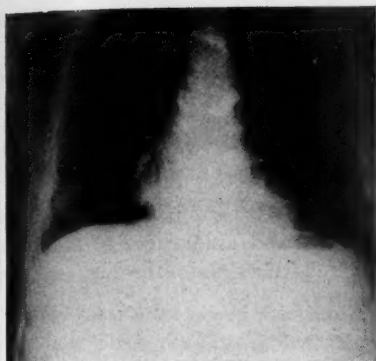
FIG. 7   FIG. 8
FIG. 7. Neurofibroma in the postero-inferior mediastinum
FIG. 8. Oblique view (same patient as figure 7) demonstrating the posterior position of the neurofibroma in the mediastinum.

Neurofibroma and neurinoma. Neurofibroma is the lesion of von Reckling-hausen's disease and originates from the peripheral nerves or endoneural connective tissue. Neurinoma, or schwannoma, develops from the nerve sheath and histologically shows fusiform cells with palisading of the nuclei. Grossly and clinically, however, they are very similar and may be considered together. These lesions ordinarily occur in the posterior mediastinum in the paravertebral gutter. They are rounded or ovoid in shape, and, occasionally, may have an hourglass appearance when there is an extension of the tumor through an intervertebral foramen. The neoplasm should be removed for the relief of symptoms and because of the possibility of malignancy either being present or subsequently developing (figs. 7 and 8).

Neurogenic sarcoma. Malignant degeneration may occur in either a neurofibroma or neurinoma. Various authors[5] have reported an incidence of malignancy ranging from 20 to 40 per cent. Peripheral invasion is common, and the prognosis usually is poor. Early excision is the only hope of a good result.

Neuroblastoma. Neuroblastoma often is seen as a retroperitoneal tumor in young children, but may occur in the mediastinum in any age group. This lesion apparently arises in the mediastinum from sympathetic nerve tissue. It is rapid-growing and very invasive. Early complete excision is indicated if there is no evidence of distant metastasis.

Ganglioneuroma and ganglioneurosarcoma. These tumors arise from the sympathetic ganglia in the paravertebral gutter. They occur more often in children and young adults, but may be present at any age. Approximately 15 per cent of these tumors are sarcomas. Early wide excision is the proper treatment.

Enterogenous Cysts

Enterogenous cysts are congenital in origin, and are located in the posterior mediastinum, more often on the right side. They are relatively uncommon, and

are discovered more frequently in infancy or early childhood. The esophagenic cyst in our series, however, was present in an adult.

Symptoms and signs of enterogenous cysts usually are due to pressure and vary with the size of the lesion. The preferable treatment is excision of the cyst and repair of any defect in the esophagus or stomach.

METASTATIC MEDIASTINAL TUMORS

Mediastinal metastasis generally is due to primary carcinoma of either the esophagus or bronchus, but may be from malignant lesions located elsewhere. Careful examination for the primary lesion often will establish the diagnosis; but, occasionally, exploratory thoracotomy may be required.

MISCELLANEOUS TUMORS

Parathyroid Adenomas

Parathyroid adenomas may occur in the mediastinum and assume particular importance when associated with hypercalcemia, hypophosphoremia, and nephrolithiasis. Parathyroid adenomas usually are benign, especially when located in the mediastinum. Surgical excision is the proper treatment.

Lipomas

Lipomas can occur in any portion of the thorax; and, occasionally, they are quite large. Roentgenologically, it may be difficult to differentiate these tumors from cysts. Associated symptoms usually are due to pressure and vary with the size of the tumor. Removal is indicated to establish the diagnosis and relieve the symptoms (figs. 9 and 10).

Blood Vascular Tumors

Tumors of blood vascular origin in the mediastinum are rare. Of this group, benign hemangiomas occur most frequently. True mediastinal endotheliomas are uncommon, and hemangioendotheliomas seldom are found.

FIG. 9   FIG. 10

FIG. 9. Lipoma in the right cardiophrenic angle

FIG. 10. Oblique view (same patient as figure 9) revealing the lipoma extending anteriorly to the anterior thoracic wall.

Symptoms from mediastinal tumors of blood vascular origin vary widely and depend largely on the size and position of the lesion. Rarely, a bruit may be present. Pain usually is due to invasion of surrounding structures by a malignant lesion. Cough, dyspnea, and hoarseness may be present if the respiratory tract or recurrent laryngeal nerves are involved.

Frequently, a good result will follow excision of a benign lesion, but the prognosis for the patient with a malignant lesion is poor. Early complete excision before metastasis occurs is the desired treatment.

Chondromas and Chondrosarcomas

Chondromas may originate from costal cartilage, the sternum, or from the vertebrae. Their position in the thorax often suggests their proper identity. Surgical excision is indicated and, when sarcoma is present, wide resection with reconstruction of the chest wall is recommended.

Mesothelioma

Mesothelioma of the pleura is a rare lesion which may involve either the visceral or parietal pleura. These tumors may be divided into two groups, 1) localized fibrous mesothelioma and 2) diffuse malignant mesothelioma.[1]

Localized fibrous mesothelioma is benign, although recurrence is common. It is frequently associated with arthritis and clubbing of the fingers. The diagnosis rarely is made preoperatively. Surgical removal of a mesothelioma is indicated. Usually, this cures the patient of the tumor, and relief of arthritic symptoms occurs.

Diffuse malignant mesothelioma is a very malignant lesion seldom curable by any form of therapy. It is important to distinguish it from localized fibrous mesothelioma as the prognosis is so different. Radical excision of a small lesion, and palliative irradiation therapy for multiple lesions is indicated.

LESIONS SIMULATING MEDIASTINAL TUMORS

This subject is too large to consider in detail, and will be presented only briefly.

Bronchogenic Carcinoma

Carcinoma of the bronchus originating near the hilus or associated with mediastinal metastasis may closely simulate a mediastinal tumor. Bronchoscopy and cytologic examinations of sputum and bronchial secretions will usually indicate the presence of the carcinoma, but, occasionally, thoracotomy may be necessary before the identity of the tumor can be established.

Carcinoma of the Esophagus

If carcinoma of the esophagus is large, and, particularly, if the malignancy has extended into the periesophageal tissues, a primary tumor of the mediastinum may be suggested on the chest roentgenogram. Clinical history, roentgenologic examinations of the esophagus, and esophagoscopy usually will reveal the presence of an esophageal neoplasm.

Aneurysm

An aneurysm may closely simulate a true mediastinal neoplasm roentgeno-
logically. Fluoroscopic visualization of pulsation in the mass is helpful, but may
not be present or may represent transmitted pulsations from the heart or large
vessels. A positive serology in a patient with a mass in the region of the aorta is
suggestive of aneurysm, particularly if a calcified plaque can be visualized in the
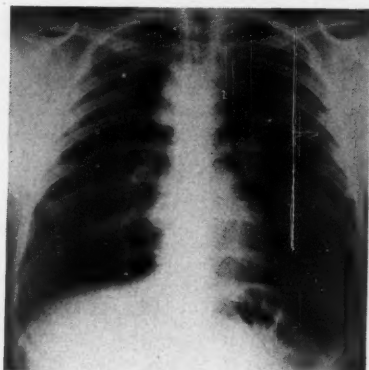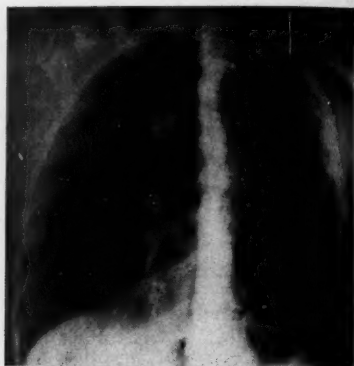
Fig. 11   Fig. 12

Fig. 11. Mediastinal tuberculoma

Fig. 12. Oblique view (same patient as figure 11) demonstrating tuberculoma in the
right superior mediastinum.
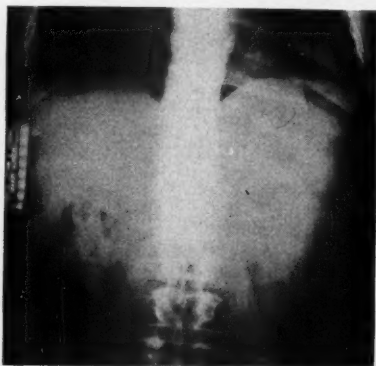
Fig. 13. Diaphragmatic hernia simulating a mediastinal tumor

lesion. Angiography and laminography have been of great aid in diagnosing these
lesions, however, exploratory thoracotomy occasionally is necessary before the
diagnosis can be established.

Boeck's Sarcoidosis, Inflammatory Lymphadenitis, and Mediastinal Empyema

Considerable difference of opinion exists concerning the pathogenesis of
Boeck's sarcoidosis, however, most writers agree that it represents a benign
lymphogranuloma. Involvement of the mediastinal lymph nodes simulating a

mediastinal neoplasm may occur. This disease produces lesions throughout the body but particularly involves the skin, lymph nodes, and lungs. Usually, the diagnosis may be established by microscopic examination of an involved lymph node.

Mediastinal lymphadenitis, occasionally, may simulate a neoplasm. Particularly is this true with the fungus diseases, tuberculosis, and the other pulmonary granulomas. The associated pulmonary or extrathoracic involvement usually will exclude a primary mediastinal neoplasm, and often the correct diagnosis can be made by a careful clinical investigation (figs. 11 and 12).

The patient with *mediastinal empyema* usually relates a history of trauma to some portion of the mediastinum or a recent intrathoracic infection. The clinical picture and roentgenographic examinations of the chest ordinarily will exclude a neoplasm. Rarely, chronic empyema will be found at thoracotomy when a neoplasm was suspected.

Diaphragmatic hernia

Diaphragmatic hernia, occasionally, may be very difficult to differentiate from a primary mediastinal tumor. This is especially true of herniations of omentum through the foramens of Bochdalek or Morgagni. Careful roentgenographic examination usually will suggest the diagnosis, but, occasionally, thoracotomy may be necessary before the lesion can be identified properly (fig. 13).

SUMMARY

As a result of annual roentgenographic surveys of the chest and better clinical investigation of patients, an increasing number of mediastinal tumors are being detected. Since many of these neoplasms are malignant or produce serious complications if neglected, early correct diagnosis and proper management are very important. A useful classification is presented, and several clinical observations which may he helpful in the recognition and management of these lesions are reviewed.

REFERENCES

1. Clagett, O. T.; McDonald, J. R., and Schmidt, H. W.: Localized fibrous mesothelioma of pleura, J. Thoracic Surg. *24:* 213 (Sept.) 1952.
2. DeBakey, M. E., and Creech, O.: Tumors of mediastinum, Texas J. Med. *46:* 588 (Aug.) 1950.
3. Harrington, S. W.: Surgical treatment in eleven cases of mediastinal and intrathoracic teratomas, J. Thoracic Surg. *3:* 50 (Oct.) 1933.
4. Heuer, G. J., and Andrus, W. DeW.: Surgery of mediastinal tumors, Am. J. Surg. *50:* 146 (Oct.) 1940.
5. Kent, E. M.; Blades, B.; Valle, A. R., and Graham, E. A.: Intrathoracic neurogenic tumors, J. Thoracic Surg. *13:* 116 (April) 1944.
6. Rusby, N. L.: Dermoid cysts and teratomata of mediastinum; review, J. Thoracic Surg. *13:* 169 (June) 1944.
7. Sabiston, D. C., Jr., and Scott, H. W., Jr.: Primary neoplasms and cysts of mediastinum, Ann. Surg. *136:* 777 (Nov.) 1952.
8. Seybold, W. D.; McDonald, J. R.; Clagett, O. T., and Good, C. A.: Tumors of thymus, J. Thoracic Surg. *20:* 195 (Aug.) 1950.

THE TREATMENT OF RECURRING ACUTE PANCREATITIS BY DECOMPRESSION OF THE BILIARY TRACT

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Recurring acute pancreatitis has become an important disease entity and its diagnosis is being made with increasing frequency as definitive studies are instituted when bizarre upper abdominal pain is encountered.

Pancreatitis well may be classified into three principle types: (1) acute pancreatitis, (2) recurring acute pancreatitis, and (3) chronic pancreatitis. Since the recurrence rate of acute pancreatitis is as high as 37 per cent in some reported series,[1] it is evident that there is considerable overlapping of types 1 and 2. It may be proper to consider that any case classified as acute pancreatitis in type 1 which recurs then more accurately should be classified as type 2. Furthermore, pancreatitis must be closely related to biliary tract disease since cholecystitis and cholelithiasis exist in 60 to 70 per cent of individuals having pancreatitis. Many cases of so-called postoperative biliary dyskinesia are actually instances of recurring acute pancreatitis, but again the pancreatitis may well result from physiologic abnormalities of the choledochoduodenal apparatus or other abnormalities of the transduodenal segment of the common duct.

The final definitive diagnosis of acute pancreatitis rests on finding elevations of the serum amylase or lipase, disturbances of the calcium content and to specific findings at the time of operative exploration. Serum amylase studies should be done as a part of the differential diagnosis of any upper abdominal discomfort which is of sufficient consequence to warrant serious study. Alterations of these diastase levels are not restricted entirely to inflammatory conditions in the pancreas but also may be due to carcinoma of the pancreas, inflammatory and obstructive lesions of the salivary glands, perforated duodenal ulcer, damage to the circulation of the bowel as encountered in strangulation and renal retention. The concentration of diastase in the urine is from two to six times that of the blood serum and, when there is an elevation of amylase in the blood, this elevation can be demonstrated for a somewhat longer period of time by increased excretion into the urine, except when the elevation is due to renal retention.

Unfortunately, significant elevation of the diastase levels are not regularly present during upper abdominal pain which is due to recurring acute pancreatitis. Apparently the diastase levels increase only when there has resulted some extravasation of the external pancreatic secretion into surrounding tissues or into the parenchyma of the pancreas from where it is absorbed in abnormal

quantities. These facts add to the difficulty of finding alterations of the serum amylase and, consequently, make the definitive diagnosis of acute pancreatitis in the more mild attacks more difficult to establish. Usually several blood amylase determinations must be made before it definitely can be demonstrated that there is a relationship between the upper abdominal complaint and the diastase level. Occasionally an attack can be precipitated by the administration of prostigmine or morphine when the gallbladder is full. In one instance, an attack of acute pancreatitis was precipitated by the oral administration of glucose during a glucose tolerance test. It is our belief that while recurring acute pancreatitis can be considered a separate disease entity, it consists essentially of repeated attacks of acute pancreatitis. Ordinarily, the recognized entity, acute pancreatitis, is a relatively severe pancreatic disturbance. The recurrence rate of this entity is reported by some authors[1] to be as high as 37 per cent. Usually the recurrence rate of acute pancreatitis is considered to be quite low.[6] This variation may simply be a difference in classification.

Since this report deals with treatment by decompression of the biliary tract, we are concerned primarily with those instances where there is a common opening between the duct of Wirsung and the transduodenal segment of the common bile duct. We shall not concern ourselves in this discussion with acute pancreatitis arising from causes other than regurgitation of bile into the pancreatic duct.

In order that there may be regurgitation of bile into the pancreatic duct, it is imperative that the pressure in the common bile duct exceed that of the pancreatic duct of Wirsung. The liver and the pancreas can both excrete against a maximum pressure of approximately 22 cm. of water. Therefore, for the pressure in the common duct to exceed that in the pancreatic duct, it must mean that either the gallbladder is contracting with a relatively high pressure against resistance at the ampullary outlet or, in the event the gallbladder has been removed or is nonfunctioning, that the liver is secreting bile at a somewhat higher pressure than the pressure in the pancreatic duct. This conceivably could exist at a time when there was a relative ischemia of the pancreas due, for example, to vasospasm of the pancreatic arteriolar bed. Local ischemia most certainly would favor pancreatic damage with resultant pancreatitis of varying degrees of severity. Another mechanism by which reflux of bile into the pancreatic ducts could occur would be in the event that the pancreatic ducts are being decompressed by direct drainage of the pancreatic secretion by way of the duct of Santorini. Ordinarily, the ducts of Wirsung and Santorini communicate. It is even conceivable that bile regularly might be finding its way into the duodenum in the presence of increased resistance at the ampullary opening by way of regurgitation into the duct of Wirsung up to its junction with the duct of Santorini and then down by this duct to enter the duodenum. With bile in this situation, it is readily conceivable that should there be a momentary ischemia of pancreatic tissues, they would be particularly vulnerable to damage.

It is interesting to recount some of the pressure relationships which exist in the normal biliary tract (fig. 1). Ordinarily, bile will pass from the common
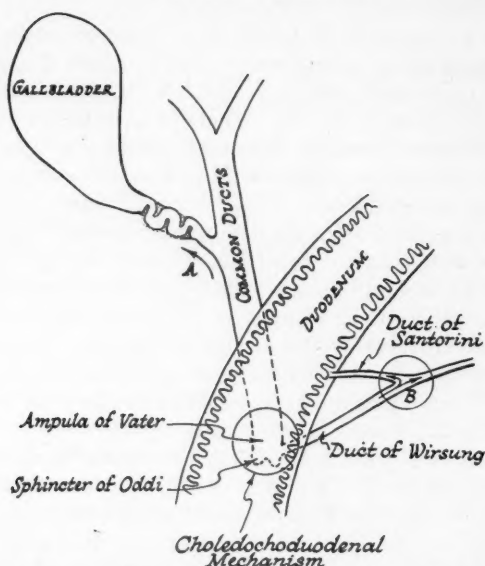
FIG. 1. Schematic representation of the hydrodynamics of the biliary and pancreatic duct systems. The gallbladder is a decompressing mechanism of small volume but large capacity with the thin liver bile entering when the pressure in common duct reaches 5 to 7 cm. of H_2O (A). The liver and pancreas can excrete against a pressure approximately equal to the arterial capillary pressure (20 to 25 cm. H_2O). The gallbladder, on stimulation, can develop a pressure in excess of 25 cm. H_2O. The choledochoduodenal mechanism can withstand a pressure in excess of 25 cm. H_2O. Physiologically the sphincter mechanism relaxes when the gallbladder is stimulated to contract. When the sphincter mechanism is in spasm, bile might reflux up the duct of Wirsung and enter the duodenum via duct of Santorini (B). External pancreatic secretions have been demonstrated to be present in 15 per cent of normal gallbladders.

ducts to the relaxed gallbladder when the pressure reaches 5 to 7 centimeters of water in the common duct. The maximum pressure levels attained momentarily when the gallbladder undergoes contractions with obstruction at the ampullary outlet is not definitely known, but it is reasonable to consider that it can become relatively high. The gallbladder, in view of its physiologic property of concentrating bile, can act as a decompressing mechanism for the biliary tract over a period of several hours in the presence of spasm of the choledochoduodenal mechanism without the development of excessive pressures in the biliary tract or regurgitation into the pancreatic system.[3] It is not unusual for pancreatic secretions to reflux up the common duct and into the gallbladder since Nash[4] reports that bile containing pancreatic secretions can be aspirated from the gallbladder of 15 per cent of normal subjects. As a consequence then, the gallbladder also functions as a decompressing mechanism for the pancreatic duct when there is a common ampullary opening between the duct systems of the pancreas and liver.

Westphal[7] made the following observations on the interrelationship between contractions of the gallbladder and tone of the choledochoduodenal sphincter

apparatus following stimulation of the vagus with: (1) weak, (2) moderate, and (3) strong faradic currents.

    1. Weak faradic current:

        Relaxation of the ampulla of Vater and contraction of the gallbladder with peristalsis of the antrum.

    2. Moderate faradic current:

        Vigorous contractions of the gallbladder, increased ampullary tone and rapid emptying of the gallbladder.

    3. Strong faradic current:

        Maximum contractions of the gallbladder and spasm of the sphincter of Oddi.

Should the gallbladder have been removed or if it should have lost its function to absorb fluid and concentrate bile as a decompressing mechanism for the duct system, then the pressure in these systems will be determined by the tone of the choledochoduodenal mechanism and the maximum excreting force of the liver and pancreas. If there has not been prior fibrotic disease in the common biliary duct system and if there is an increased tone of the choledochoduodenal mechanism, then this duct system will become dilated because of the prolonged increased pressure.
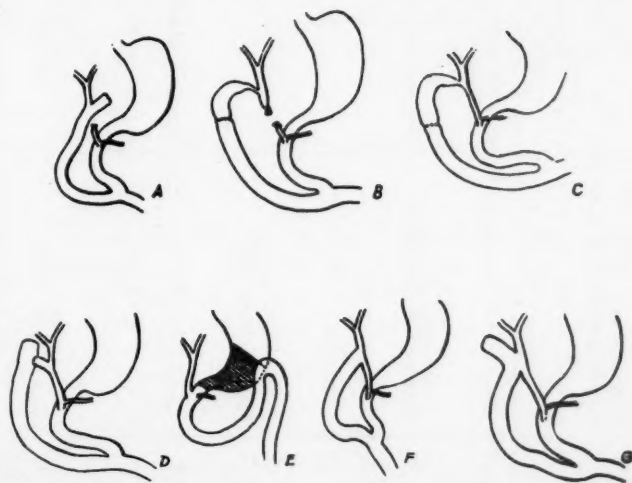
Fig. 2. Schematic representation of the various methods which are used in the application of the Roux Y technic for decompression of the biliary tract on patients reported in this article.

A. Whenever the end of the common duct is anastomosed to the jejunum, the duct should enter the side of the bowel in preference to the end because of lessened tendency of stricture formation. If the stump of the duct is sufficiently long, it should be split up one side and anastomosed to the end of the defunctionalized limb of the Roux Y.

E. Presents the procedure when choledochoduodenostomy has been done previously. Gastric resection with an ante colic gastrojejunostomy establishes essentially a defunctionalized loop of the Roux Y principle (See table 2.) The defunctionalized limb of the Roux Y should be a minimum of 18 inches long.

TABLE 1

	Patient	Age	Sex	Previous Operations	Type of Decompression See fig 2 (A-G)	Date of Operation	Remarks
1	L. A.	49	M	Mastoidectomy 1918 Tonsils and adenoids removed in 1933	A§	10/29/49	Preoperative: First attack Apr. 1948, Serum amylase 1050 U; 2nd attack Aug. 1949, Serum amylase 1000 U; 3rd attack Oct. 15, 1949, Serum amylase 650 U Postoperative: Two attacks of upper abdominal pain with chills and fever, April 1951 and March 1952. Asymptomatic thereafter.
2	L. R.	41	M	Hemorrhoidectomy 1934	Finney pyloroplasty C	7/13/51	Preoperative: Serum amylase May 10, 1951, 512 U; nine attacks beginning in May 1948. Postoperative: Occasional mild dyspepsia. Result satisfactory.
3	M. N.	60	M	Cholecystectomy Appendectomy 1928	D	2/11/52	Acute attack precipitated by glucose tolerance test. Serum amylase 680 U. Asymptomatic since operation.
4	S. G.	44	F	*	G	12/17/52	Asymptomatic since operation.
5	M. H.	22	F	†	F	2/13/53	Postoperatively patient became pregnant with morning sickness and vomiting. Serum amylase normal.
6	E. O.	31	F	Appendectomy 1940	C	3/20/53	Cholecystitis and cholelithiasis. Proved common ampulla by cholangiogram. Serum amylase 516 U, 3/6/53. Asymptomatic since operation.

7	W. W.	52	M	Appendectomy, hemorrhoidectomy		C	4/29/53	Cholangiogram shows common ampulla. Asymptomatic since operation.
8	M. M.	35	F	Tubal ligation		C	5/23/52	Diagnosis of pancreatitis at operation. Postoperative attack upper abdominal pain. Amylase 250 u, May 27, 1953. Splanchnic block gave relief.
9	I. S.	57	F	None	Appendectomy	C	6/ 8/53	Common ampulla proved. Chr. alcoholic—cirrhosis liver diagnosed 1948. Gallbladder and common duct normal.
10	F. D.		F	‡	Plication	C	7/24/53	Asymptomatic since operation.
11	C. P.	48	M	Cholecystectomy 1947		F	3/30/53	Operation poorly done technically. Stormy postoperative course. Now asymptomatic.
12	W. K.	42	M	Gastrectomy 3/22/49		C	3/ 6/50	Chronic pancreatitis. Asymptomatic since operation.

* Appendectomy 1922; tubal ligation—uterine suspension 1924. Cholecystectomy, exploration common duct insertion T tube 1950. Stormy postoperative course. Had attacks of upper abdominal pain at about two weeks intervals since cholecystectomy. Serum amylase 340 U Nov. 27, 1952. Serum lipase 565, Nov. 28, 1952. Blood sugar 195, Nov. 28, 1952. Xanthomatosis—blood cholesterol Dec. 1, 1952, 445 mg. per cent. † Appendectomy 1947. Cholecystectomy 1950. Partial gastrectomy 1952. Initial diagnosis of acute pancreatitis Aug. 25, 1951. Serum amylase 566 U.

‡ Appendectomy 1937, operations for adhesions twice in 1942 and twice in 1950, exploratory laparotomy 1952—patient jaundiced, biopsy of pancreas, exploration of common bile duct, sphincterectomy. Diagnosis: chronic pancreatitis, tale granuloma, adenoma of the pancreas. Had repeated elevations of serum amylase.

Operation July 24, 1953 included exploratory laparotomy, freeing of extensive adhesions followed by Noble plication, Roux Y cholecystoje-junostomy. Pancreas normal—no evidence of tale granuloma.

§ Procedure similar to technique used by Bowers, R. F.; Surg, 30 116–129 (1951).

TABLE 2

Use of Roux Y principle to eliminate regurgitation following choledochoduodenostomy

Patient	Age	Sex	Date of Gastric Resection	Remarks
R. C.*	53	M	7/28/51 (Fig. 2-F)	Patient has calcifications in pancreas. There has been some improvement. Cooperation poor—difficult to evaluate. Result not satisfactory.
J. Y.†	26	F	9/12/51 (Fig. 2-F)	Poor technical procedure at operation. Short loop gastroenterostomy done. Regurgitation not cured. Result—failure.
E. N.‡	48	M	9/21/51 (Fig. 2-F)	Complaint of abdominal pain for first month postoperative. Well thereafter, gained 35 lbs. Regurgitation corrected.
M. C.§	52	F	10/19/51 (Fig. 2-F)	Completely well.

* *Previous operations:* Perforated duodenal ulcer—closure 1940, appendectomy 1946; choledochoduodenostomy September 6, 1946. Diagnosis: chronic recurrent pancreatitis with lithiasis. Cholangitis with chills and fever followed choledochoduodenostomy. Biliary tree filled with air and visible on roentgenogram.

† *Previous operations:* Cholecystectomy 1948, exploration 1949, stones removed from common duct with T tube drainage. Continued right upper quadrant pain, choledocho-duodenostomy September 11, 1950, delivery May 21, 1951 with acute catarrhal jaundice (?). Recurrent chills, fever and jaundice. September 12, 1951 gastrectomy with short looped, retrocolic, gastrojejunostomy. Re-explored October 24, 1951—bile peritonitis, ruptured duodenal stump, dilitation of proximal loop with obstruction at anastomosis. Entero-enterostomy to decompress proximal loop. Chills and fever have continued and the biliary tree is still outlined by air.

‡ *Previous operations:* Perforated ulcer 1928, cholecystectomy 1941; continued to have pain, jaundice, exploration for adhesions in 1945, abandoned because of patient's condition. 1948: stones removed from common duct with catheter drainage of common duct, freeing of adhesions and ileoileostomy. Pain, jaundice, chills and fever continued intermittently. September 18, 1950, Choledochoduodenostomy, pyloroplasty. Following this procedure, the patient had nausea, vomiting, pain and hematemesis. On August 14, 1951, it was demo-strated by roentgenogram that biliary tree was filled with air and barium refluxed into common duct. September 21, 1951: Gastrectomy, Hoffmeister, long loop, ante colic. Es-sentially well, no reflux demonstrable, weight gain from 108 to 145 pounds.

§ Hysterectomy; cholecystectomy and gastroenterostomy followed by immediate post-operative jaundice August 1, 1943. Choledochoduodenostomy September 10, 1943. Begin-ning in July 1951, right upper quadrant pain, intermittent jaundice, chills, fever, nausea and vomiting. Roentgenogram showed air in biliary tree September 4, 1951. October 19, 1951: gastric resection, long loop, ante colic. Asymptomatic since operation with gain of normal weight.

When it can be demonstrated that obstruction to drainage of bile does not exist following choledochoduodenostomy and that regurgitation does exist, the condition can be satis-factorily corrected by a gastric resection and a long loop gastroenterostomy applying es-sentially the Roux Y principle.

TREATMENT OF RECURRING ACUTE PANCREATITIS

An acute attack of recurring acute pancreatitis should be treated as any other attack of acute pancreatitis of comparable severity. At a later date treatment should be directed towards insuring continuous decompression of the biliary tract to prevent the recurrences characteristic of this entity. Treatment of an acute attack presents a very nicely balanced physiologic problem. Every effort should be made to decrease stimulation of pancreatic secretion and to increase relaxation of the sphincter apparatus without interfering with the blood supply to the pancreatic tissues.

Popper[5] has outlined the principles of treating acute pancreatitis quite adequately. Gastric suction and nothing by mouth immediately should be instituted. Demerol is preferred to morphine for the control of pain. One of the parasympatholytic drugs, such as Banthine, 100 mg. every six hours, should be administered parenterally. Fluids and electrolytes should be administered by intravenous injection. Care must be taken in the intravenous administration of glucose since these patients tend to have mild diabetes, and an increase of glucose intravenously has some cholinergic action. The use of insulin and glucose given intravenously must be very carefully controlled. Blood sugar levels should be maintained at relatively normal values, because gastric secretion may be stimulated by hypoglycemia via the vagus mechanism. This mechanism will, to a degree, be blocked by the administration of parasympatholytic drugs. Bilateral splanchnic blocks should be instituted early and repeated as often as indicated to control pain.[2] Very likely, this measure is of particular value in that it prevents vasospasm in the arteriolar bed of the pancreas, thereby maintaining a good blood supply to the parenchyma of the pancreas rendering it less susceptible to damage. Frequently, the attacks of recurring acute pancreatitis are so mild and of such short duration that they require no definitive treatment. In any event, treatment directed towards permanent decompression of the biliary tract is not undertaken during the period when acute inflammatory reaction involves the biliary tract.

We have used all of the operative methods indicated in figure 2 for decompressing the biliary tract by use of the Roux Y principle. Our first patient was operated upon in October 1949, using the procedure illustrated in figure 2-A. Subsequently, we advocated the procedure illustrated in figure 2-C whenever the gallbladder was present and contained normal bile. With increased experience, we believe that the method of choice is that illustrated in figure 2-F. If the gallbladder is present, a cholecystectomy is done, and the common duct is opened and explored. Usually a stone is not encountered in the common duct, but quite frequently there is resistance to passing a probe into the duodenum. Obstruction at the ampulla of Vater may be from several different causes. There may be fibrosis with narrowing of the outlet, or the resistance may be purely one of physiologically increased spasm which usually cannot be detected when the patient is under anesthesia.

The details of the operative procedure indicated in figure 2-F are clearly
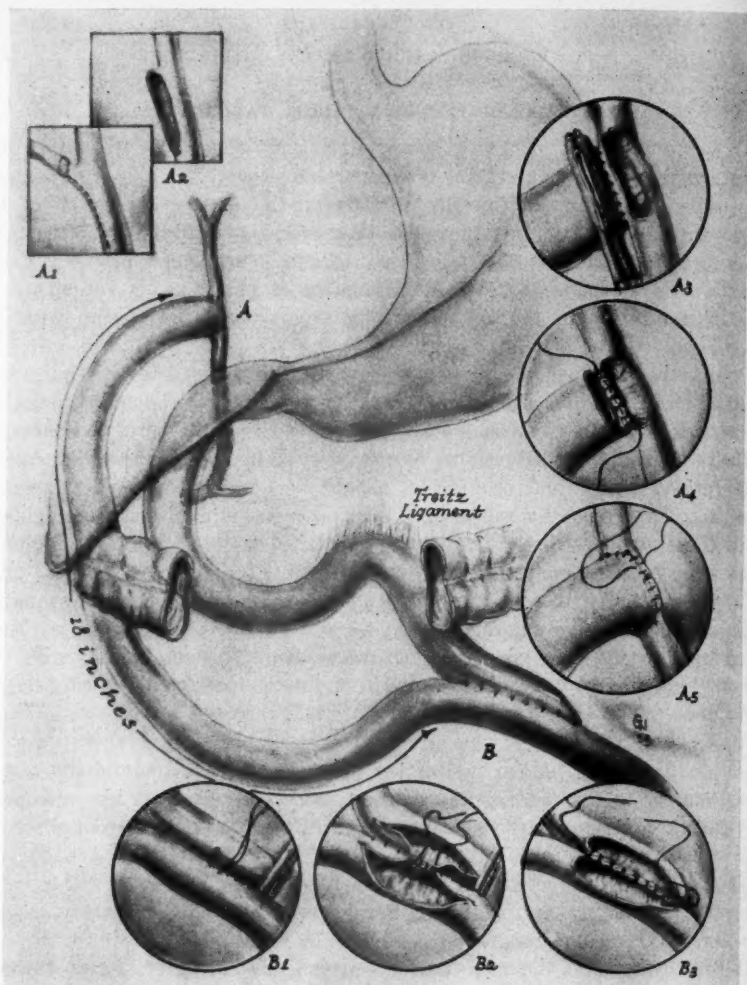
FIG. 3. Details of the choledochojejunostomy Roux Y procedure indicated in figure 2-F.
A. Anastomosis of the end of the defunctionalized loop of jejunum. This loop should be
18 inches long and is usually placed retrocolic to the side of the common duct.

A_1 After cholecystectomy, the common duct is split sufficiently to receive the end of
the jejunum.

A_2 The general appearance of the ostium into the common duct. The stump of the
cystic duct constitutes part of the opening.

A_3 Showing the posterior interrupted row of sutures of the choledochojejunostomy
in place.

A_4 The continuous inner mucosal suture of no. 000 chromic catgut introduced poste-
riorly. It will be continued anteriorly as an inverting suture.

A_5 The completed choledochojejunostomy.

B. Lateral end-lateral jejunojejunostomy. (Poth, E. J., S. G. & O. *91:* 656–659 (1950).)

B_1 Posterior outer row of interrupted no. 0000 braided nylon suture being placed.

B_2 Two segments of jejunum have been opened and the posterior row of continuous
no. 00 chromic catgut introduced.

B_3 The proximal segment of jejunum has been trimmed and the anastomosis being
completed.

The choledochojejunostomy as done in this procedure delivers the bile into the jejunum
and interrupts the flow of bile through the intraduodenal segment of the common duct.
Should the ampulla of Vater become obstructed, pancreatic secretions will reflux up the
common duct to enter the jejunum.

Obstruction of the common duct at the site of the anastomosis is unlikely because the
line of suture does not encircle the duct.

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presented in figure 3. Particular emphasis should be placed on two advantages of this procedure: (1) the common duct is not encircled by the anastomosing suture although the continuity of the duct is effectively severed in so far as biliary drainage is concerned, and (2), pancreatic secretions can reflux up the proximal segment of the common duct to enter the jejunum should the ampullary opening become occluded.

Results of Treatment of Recurring Acute Pancreatitis by Decompression of the Biliary Tract by Use of the Roux Y Jejunostomy

Table 1 summarizes the cases treated by the procedure. The results have been quite satisfactory. There have been no frank failures.

It appears that the results are better when the end of the jejunum is anastomosed to the gallbladder than when the common duct is divided and its end implanted into the side of the jejunal loop probably because of better drainage obtained by the cholecystojejunostomy. However, in case no. 8 (M. M.) the patient had an attack about a year after cholecystojejunostomy, and it may be that the cystic duct had become occluded. Because of the experience gained by application of the principle of internal drainage in the treatment of biliary tract disease proper, we now believe that the procedure presented in figure 3 is preferred. Ordinarily, the patients have a smooth, uncomplicated postoperative convalescence.

DISCUSSION

The fundamental concept here developed for treatment of recurring acute pancreatitis is to establish continuous decompression of the combined duct systems of the liver and pancreas by permanent internal drainage whereby the normal hydrodynamics is largely restored with arrest of anatomic and physiologic aberrations. Decompression by means of the Roux Y technic applied as an anastomosis of the end of the jejunum to the side of the common bile duct gives the best assurance of a lasting result. A long loop of jejunum will safeguard against infection by regurgitation of bowel contents into the biliary and pancreatic duct systems. It is conceivable that the ampulla of Vater could be incised and the edges stitched into the incision to form an opening sufficiently large to overcome subsequent stricture and at the same time be sufficiently small to prevent regurgitation of duodenal contents into the duct system, but the surgeon cannot consistently reproduce such an opening. The failures seemingly would be prohibitively high.

Transplantation of the pancreatic duct to an individual site in the duodenum is possible in selected instances but is technically difficult.

The use of an end to side Roux Y (end of jejunum to side of common duct) with a sufficiently long loop of jejunum will decompress the duct system without destroying the common bile duct and have little likelihood of stenosis since the line of suture does not encircle the relatively small common duct. Furthermore, should the ampullary opening become obstructed, pancreatic excretions can reflux up the intraduodenal segment of the common duct to enter the jejunal

loop. So long as free drainage is maintained and particulate matter is not forced into the ducts, there is little danger of cholangitis.

One of us (E. J. P.) has, on several occasions, established internal drainage of the biliary tract, using the Roux Y procedure in the presence of long standing infection and scarring of the biliary ducts, with excellent results. For example, a 36 year old white man had been operated upon elsewhere two years previously. The tissues were so involved in scar and active inflammation that only chole-cystostomy was done to remove stones and a T tube inserted into the common duct. The T tube was still in place and draining bile freely. A choledochogram showed an extensively scarred common duct; tortuous and segmented in outline. After washing the system with 10 cc. of ether daily for three months, the T tube was removed. Within six weeks, the patient again had fever, pain and anorexia. He continued to have pain, occasional fever and jaundice and anorexia for about a year before he consented to re-exploration on July 17, 1952. The gall-bladder and common duct were thickened. The common duct was opened and a stone 1 by 2 cm. was removed. The ampulla of Vater was dilated widely. The gallbladder was opened, no stones were present and normal colored, clear bile was in the gallbladder. A Roux Y cholecystojejunostomy was made and the incision in the common duct was closed. The abdomen was closed without drainage by repair of a postoperative ventral hernia. The wound healed per primam.

The patient has returned to work; has continued afebrile and free of pain; has gained 20 lbs. in weight, and considers himself well. Apparently infection will subside with satisfactory anatomic restoration when adequate continuous drainage of the duct system is established.

There has been no frank failure following this method for the internal drainage and decompression of the duct system in the treatment of recurring acute pancreatitis. Not every patient has had an entirely uncomplicated postoperative course. The earliest patient of this series, L. T. A. (no. 1) table 1, (Oct. 1949) was treated as indicated in figure 2-*A*. Prior to operation he had attacks of severe upper abdominal pain every two to three weeks. He has had two attacks of upper abdominal pain with chills and fever 6 and 17 months postoperatively. Thereafter, he has remained asymptomatic.

W. K., no. 12, table 1, not a case of recurring acute pancreatitis, is included because he developed an upper abdominal mass 10 days after 3 specimens for biopsy were taken from the head of the pancreas. The mass resolved spontaneously after draining pancreatic juice for three weeks. It is recommended that the pancreas not be biopsied because of danger of cyst formation.

M. M., no. 8, table 1, had an acute episode of upper abdominal pain May 27, 1953 with a serum amylase level of 250 units. The pain was relieved by splanchnic block. A cholecystojejunostomy (Roux Y) had been done May 23, 1952 to decompress the biliary tract. The decompression may no longer be effective because of possible stricture of the cystic duct. Should another attack occur, exploration will be done with the expectation of doing a cholecystectomy and an anastomosis of the end of the jejunum to the side of the common duct.

It is because of this source of failure and the possibility of the distal segment of the common duct becoming filled with debris when a cholecystojejunostomy is done that the end to side choledochojejunostomy Roux Y procedure is favored. While there is preservation of continuity of the common duct anatomically, it is interrupted in so far as the drainage of bile is concerned.

SUMMARY

Some of the anatomic and physiologic characteristics of the duct systems of the liver and pancreas are discussed in the light of their importance in the occurrence, diagnosis and treatment of recurring acute pancreatitis.

A procedure for the continuous internal drainage and decompression of these duct systems is presented and discussed.

A series of 11 cases of patients treated by the procedure is reported. Although the number of cases is small and the follow-up period short, the results seem to justify continued use of this procedure which most nearly preserves the original anatomic and physiologic relationships and may possibly correct the underlying abnormal process.

REFERENCES

1. Edlund, Y.: Acute necrosis of pancreas; analysis of clinical material, Acta chir. Scandinav. *99:* 497, 1950.
2. Gage, M., and Gillespie, G.: Acute pancreatitis and its treatment, South. M. J. *44:* 769 (Sept.) 1951.
3. Ivy, A. C.: Physiology of gallbladder, Physiol. Rev. *14:* 1 (Jan.) 1934.
4. Nash, J.: Surgical Physiology, Springfield, Ill., Charles C Thomas, 1942, p. 186.
5. Popper, H. L.: Acute pancreatitis; evaluation of classification, symptomatology, diagnosis and therapy, Am. J. Digest. Dis. *15:* 1 (Jan.) 1948.
6. Probstein, J. G.: Acute pancreatitis, J. Internat. Coll. Surgeons *15:* 147 (Feb.) 1951.
7. Westphal, K.: Motility, nerves and pathology of bile ducts, Ztschr. f. klin. Med. *96:* 22 (Jan.) 1923.

MANAGEMENT OF SURGICAL INCISIONS OF THE ABDOMEN*

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Complications in wound healing may mar an otherwise brilliant operative procedure. The incisional hernia that is a residual of poor wound healing may be more disabling than the condition for which the original operative procedure was done. On occasion a severe wound infection or a wound evisceration may be directly or indirectly responsible for the patient's death. It has been my observation in the past that there often is an unnecessarily high incidence of wound complications on teaching and charity services where the care of and the opening and closing of abdominal incisions is left to the junior surgeon. I believe that is strong evidence that too little importance is attached to wound management and too little attention is given to the subject in our training programs designed for the education of surgeons. Many of our otherwise well trained young surgeons must learn good wound management by trial and error whereas a little more effort expanded in that direction during their training period would save them and their patients much later trouble. I am sure that no one will take issue with the statement that it is of utmost importance that the surgeon be cognizant of and practice methods that insure sound healing of abdominal incisions.

Infection, dehiscence or actual evisceration, poor healing, and the residual incisional hernias are the undesirable sequelae of surgical incisions. In order for bacteria to propagate and cause clinical infection they must have cultural media to grow in and must be able to over-power the defensive mechanisms of the host. It is perhaps true that occasionally exceedingly virulent bacteria are encountered in the surgical theater and if they gain access to wound clinical infection will supervene regardless of the condition of the wound. However, from a practical standpoint, in order for bacteria to gain a foothold in a clean surgical incision, culture media of devitalized tissue, serum, or blood clot must be provided. Neighboring areas of diminished blood supply are readily accessible to inflammation spread when bacteria have gained a foothold.

It is perhaps occasionally instructive to see a surgeon who hardly rinses his hands for a scrub and then proceeds to the operation with a minimal preparation of the operative field and yet rarely produces anything less than a strong primarily healed wound. On the other hand we may occasionally see an operating room where the most meticulous antibacterial technic is carried out and yet surgeons may be plagued with wound infections. I do not wish to minimize the importance of the most rigid antiseptic procedures nor do I wish to condone straying from the accepted procedures of preoperative preparation, but I do want to stress the importance of the surgical technic in the prevention of wound infection.

It seems hardly necessary to comment that in order to produce rapid and strong wound healing previously divided tissues should be reapproximated in their normal positions. These reapproximated tissues should be traumatized as little as possible and their vascularity should have a minimum of disturbance. The practice of widely separating each layer as the incision is developed can only be condemned. It makes a pretty picture but it certainly diminishes the blood supply and viability of the tissues which can only delay and interfere with proper healing. Demonstrative dissection should be relegated to the anatomy laboratory; surgery should be done in the operating theater. The approximated wound edges should be sustained with suture material that gives sufficient support for the healing period, but yet causes a minimum of interference with the healing process. It is of course understood that the chemical and nutritional state of the body tissues is of utmost importance in relation to the healing process. However, that phase of the problem is not a part of this presentation.

With the above principals in mind a discussion of the actual opening and closing of the abdominal incision is in order. We believe the preparation of the skin of the operating field deserves comment. From my experience and that of many others I have concluded that a soap and water scrub provides the most nearly clean operative field. It is our policy to use soap and water just before the operation. A field of operation prepared the evening before may be contaminated by morning and the bacteria of the skin reduced by the procedure may have had time to gain a new foothold. The preparation is usually completed by the application of one of the commercial antibacterial agents. However, we believe that this latter step is of far less importance than the mechanical cleaning.

In the actual making of the incision it is most important to make a clean cut with as few strokes as possible. Ideally one would cut to the deep fascia in one stroke. Each secondary stroke devitalizes extra bits of tissue the size and amount, of course, depending upon the raggedness of the line of incision. As mentioned above, we see no advantage and much harm from the common practice of widely separating the subcutaneous tissues from the deep fascia. Such practice devitalizes the fascial edge and subsequently may be responsible for delayed healing of that layer. It also leaves potential pockets between the subcutaneous tissue and fascia where serum, clots, and devitalized tissue may collect and form the culture media for invading bacteria as well as to interfere with the process of healing. These principals of course apply to the further development of the incision through the remaining layers. Extensive dissection of the layers makes a beautiful picture but the dictum "operate in the operating room and save dissection and anatomic demonstrations for the anatomy laboratory" pays real reward in the subsequent healing of the wound.

Control of the bleeding from the wound edges deserves special comment. We should neither fill the incision with hemostatic ties nor should we neglect bleeding points to the extent that the closed incision is potentially filled with clots and serum. Conforming to the principle that the majority of the minute bleeding points in the subcutaneous tissues will seal off spontaneously, it is our practice to temporarily control the oozing from the wound edges with gauze pads placed

behind the fingers of the operator and his assistant until at least the deep fascia is reached. The larger vessels may be grasped before or immediately after they are cut. By this method not only will considerable time be saved, but it will be found that the majority of the small bleeding points will retract and that a minimal of clamping and tying will be necessary. It seems superfluous to mention that only the tip of the bleeding vessel together with none or as little of the surrounding tissue as possible should be grasped. Everything distal to the tie will become necrotic, and of course, it is easy to see why the wound made by the careless surgeon, who strangulates a large mass of tissue with each vascular ligation, will be a ready focus for infection and delayed healing. We believe the choice of material for vascular ties is of secondary importance. Our only objection to catgut is that some surgeons accept it as a license to use it carelessly without regard to the amount of tissue ligated. They seem to think that just because catgut is absorbable it can do no harm. On the other hand nonabsorbable sutures call for a meticulous technic which necessarily produces good results. We believe that one can do equally as well with either suture. Our preference has been for fine cotton for vascular ligatures.

Assuming that the physiologic healing processes of the patient are at or near normal, the object of wound closure should be to oppose the corresponding tissue layers and to hold them in apposition against any of the stresses or strains that may be placed upon them until firm healing has taken place. The method of closure must be such that no material will be introduced whose foreign body reaction will be enough to significantly interfere with wound healing or promote infection. Tissue edges must not be compressed enough by sutures so as to materially interfere with their viability.

Abdominal wounds, especially those of the upper abdomen, can be subjected to enormous stresses and strains during the healing period. Distension and coughing are most trying to the wound integrity. We know of no method of un-reinforced individual layer closure that provides sufficient strength to the healing wound undergoing unusual stresses, especially in the face of delayed healing due to either technical difficulties or interference with the physiologic processes. We are, therefore, of the opinion that all upper abdominal incisions, as well as those in the midabdomen and lower abdomen where there is danger of delayed healing or unusual stress, should have some reinforcement of the usual layer closure if the incidence of dehiscence and hernia are to be kept at a minimum. Originally it had been our habit to use removable silk or silk worm gut retention sutures for this purpose. These were not entirely satisfactory in view of the fact that they often were responsible for considerable pain and often were the site of troublesome stitch abscesses.

Following the reports of Babcock,[1] Jones,[2] Large,[3] Wiley[4] and others, we first resorted to the use of stainless steel wire for suture material in 1947. We soon concluded that it was the most satisfactory suture material for abdominal wound closure when either stress or delayed healing is of any significance. Subsequently, we have used it routinely for closure of all wounds of this type by a method to be described below. Because of our complete satisfaction with this

method we wish to present a detailed report of our experience with it in abdominal wound closure.

This series consists of 300 consecutive abdominal wound closures made as described below with stainless steel wire sutures. Both no. 32 and no. 30 wire were used, but our personal preference, in contradistinction to many others, has been for the finer material. In infants and young children wire as light as no. 36 was used. These cases included all upper abdominal wounds and others where it was thought that delayed healing or unusual stress might be a factor. In the usual pelvic case and benign disease of the lower abdomen it was believed that individual layer closure with cotton was satisfactory. Table I lists the various procedures in which stainless steel wire closure was utilized.

TABLE I   Distribution of Abdominal Wire Closures

Biliary Tract	132
Stomach and Duodenum	92
Intestinal Tract and Rectum	41
Miscellaneous Abdominal	22
Infants and Children	10
Splenectomy	3
Total	**300**

The method of wire closure was essentially that described by Jones[2] and other authors with some personal modifications. In contradistinction to these other authors we have preferred to close the peritoneum as a separate layer with a running suture of chromic catgut rather than to incorporate that structure in the wire sutures. We believe that the peritoneum adds very little tensile strength to the closure, and that by closing it separately there is less chance of unwittingly incorporating intra-abdominal structures between the margins of the wound. Also by this method we can much more rapidly seal the abdomen off from the outside environment. The deep fascia, the muscle, and the preperitoneal fascial tissues are incorporated in figure of eight stitches as illustrated in figure 1. The first bite is taken well back from the cut edge through the fascia, muscle and preperitoneal tissues. It is carried across to the other side and out through the same layers. A second loop is taken which more accurately approximates the anterior fascial edges. The suture is carefully tied with a triple throw square knot. The wires are grasped with a forceps distal to the knot. The free ends are cut flush with the forceps which is then rotated so that the sharp ends are pointed inwards. It is most important to tie the wire loose enough so that the tissue edges are barely approximated. Then there will be less interference with the viability of the tissues. Furthermore, early in our experience with the use of wire it was common for the patient to complain bitterly of spasms of pain in the incision for the first few days postoperatively. This has been practically eliminated since we have used special care not to tie the wire sutures too tightly. These wire sutures are usually placed about 1 inch apart. In most incidences we com-

plete the approximation of the anterior fascia with a few interrupted stitches of cotton placed between the figure of eight sutures. The skin is closed with interrupted stitches of cotton. We believe that the added time necessary for this type of suturing, as compared to the time necessary for the running stitch, is adequately compensated for. Intermittantly throughout the closure, the wound is repeatedly irrigated with copious quantities of physiologic sodium chloride solution. We believe that this mechanical cleansing washes out bits of devitalized tissue, clots, bacteria and other foreign debris and materially reduces the chances of wound infection.

We have divided wound complications into major and minor catagories. Major complications include dehiscence or rupture, clinical infection or drainage which prolongs the recovery period or necessitates further operative procedures, and hernia. Minor complications include stitch abscesses, infection about drainage sites, and small subcutaneous collections of serum or blood clots which in no way interferes with the expected progress of convalescence.
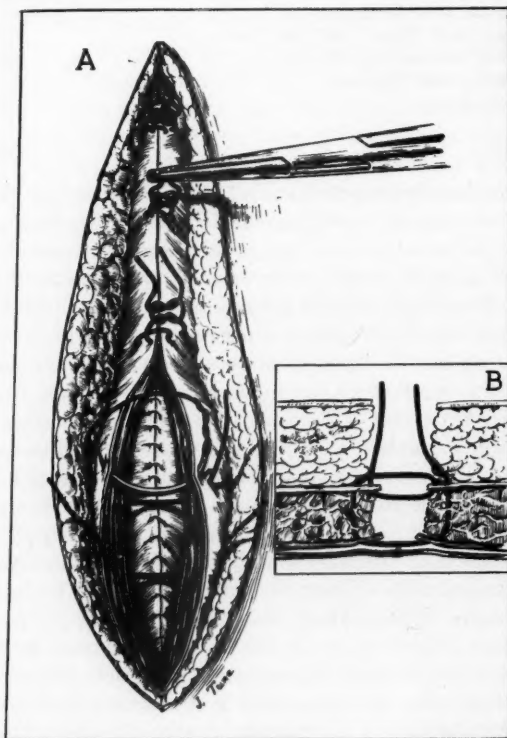
FIG. 1. Wire closure of the abdominal wall. *A*. The peritoneum has been closed with a running suture of chromic catgut. The successive steps in inserting and tying the wire sutures are shown from below upwards. *B*. Cross section showing wire inserted and ready for tying. The deep loop should be placed approximately 1 inch back from the wound edge.

There were no postoperative wound ruptures or dehiscences in this series. There was no incidence of major infection. The same method of closure was used in all age groups from the first day of life into the ninth decade. No attempt was made to protect the wounds and all patients were ambulatory as soon as possible, usually the day following operation. The only known subsequent hernia followed an incision made for cholecystectomy. The patient in whom this occurred developed postoperative pneumonia accompanied by a severe persistant cough and a troublesome paralytic ileus with marked abdominal distension. No accurate record was kept of minor complications but they were not impressive. In a few cases of cholecystectomy in which drains passed through the lower ends of the incisions it was necessary to evacuate small purulent collections by probing in the drainage tracts. On a few occasions it was necessary to remove individual wire sutures which were responsible for pain and soreness. These usually were in very thin individuals in whom the wire readily could be felt through the intact skin. As a rule these were removed with local infiltration anaesthetic, usually as a dressing room procedure. In 2 cases it was considered necessary to remove all of the wire sutures. In no case was a wire suture responsible for a draining sinus. As far as we could determine wire was in no way responsible for any delay in healing.

SUMMARY

We have presented rather briefly a discussion of the practical aspects in the management of abdominal surgical incisions. It is our opinion that in many of the surgical teaching programs too little attention is given to the importance of the wound and that consequently many young surgeons start in their practice with an unnecessary high incidence of complications in their incisions. If they had been more properly grounded in that phase of their work, both they and their patients would have benefitted.

We have presented our experience with stainless steel wire as a suture material. Although the wire suture technic of closure necessarily takes a little more time than others, we believe that the low incidence of complications and the rapid solid healing more than pays both the patient and the surgeon for the little added effort.

REFERENCES

1. Babcock, W. W.: Symposium on modern trends in surgery; metallic sutures and ligatures, S. Clin. North America 27: 1435 (Dec.) 1947.
2. Jones, T. E., Newell, E. T., Jr., and Brubaker, R. E.: Use of alloy steel wire in closure of abdominal wounds, Surg., Gynec. & Obst. 72: 1056 (June) 1941.
3. Large, O. P.: Comparison of tissue reactions from new sutures, Am. J. Surg. 60: 415 (June) 1943.
4. Wiley, H. M. and Sugarbaker, E. D.: Evaluation of abdominal wound closure, Surg., Gynec. & Obst. 85: 81 (July) 1947.

DIVERTICULA OF THE URETHRA IN THE FEMALE

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The clinical entity, diverticulum of the urethra, has been recognized for many years. A review of the history of this disease shows that it was reported in detail as early as 1869 by Priestley,[5] who recorded 3 cases as *examples of cyst formation in the neighborhood of the female urethra.* Since that time, numerous reports have appeared in the literature under such pseudonyms as saccular dilatation of the urethra, urethrocele, urine pocket, prolapse of the urethra, herniation of the urethra and urethral abcess. In the past decade there has been a growing awareness by physicians of this condition, but textbooks of pathology have been singularly free of references to it. Clinical textbooks likewise have failed to give space commensurate with it's importance.

The purpose of this paper is to call attention to certain salient facts about diverticula of the urethra. First, the condition is discovered in almost direct proportion to the frequency with which it is searched for and considered in diagnosis. This has been amply stressed by Carson,[1] Neel,[4] Moore[3] and others. The personal files upon which this report is based, show that no case of diverticulum was recognized from 1946 to 1950. In contrast, from 1950 to 1953, during which time a careful search for diverticulum was made on each female patient, 9 cases were found. A review of the case histories at Colorado General Hospital from 1930 to 1950 showed but three urethral diverticula, yet this is a large institution with a busy out-patient service.

Second, nearly all patients give a history of long standing trouble with the urinary tract. As stressed by Wharton,[7] the most common symptoms are frequency, burning, pain and incontinence. In many instances the history carries back for 10 to 20 years. One of our patients had had trouble for 32 years, and all but 2 had had symptoms for more than 10 years. All of our patients had had previous urologic treatment consisting of urethral instillations or dilations. All but 2 had had previous cystoscopic examination and 1 insists that she was cystoscoped at least 50 times and had been examined by 13 different urologists.

A third fact worth stressing, is that many of these patients complain of an almost pathognomonic symptom. They leak urine for a short time after voiding. This is due to the gradual emptying of the diverticulum which had been filled during the act of urinating, and most often is seen when the diverticulum has a good sized opening into the urethra. One of our patients exhibited this phenomenon to such an extent that her only complaint was terminal incontinence. To correct this distressing condition, she had submitted to a hysterectomy, and a year later to repair of a so-called cystocele. Eight of our 9 patients complained of urinary leakage in greater or less degree. The only patient who did not leak urine had a diverticulum which could not be emptied and which acted as an urethral obstruction. At operation, the diverticulum was found to be filled with a tumor mass.

TABLE 1

Frequency of the signs and symptoms which lead to a presumptive diagnosis of diverticulum of the urethra. All patients had had recurrent attacks of dysuria

Patient	Age	Duration of Symptoms	Leakage of Urine	Mass	Tender Urethro-Vaginal Area	Secretion Obtained on Urethral Massage
		yr.				
C. T.	64	2	No	+	+	+
M. B.	52	32	Yes	0	+	+
L. S.	60	10	Yes	0	+	+
B. H.	32	13	Yes	+	+	0
D. W.	44	3	Yes	+	+	+
R. J.	72	15	Yes	+	+	+
E. K.	76	13	Yes	+	+	+
J. Z.	37	19	Yes	0	+	+
J. W.	40	10	Yes	0	+	0

Finally, many patients will exhibit one or more of three clinical signs which strongly suggest urethral diverticulum. One sign is the presence of a mass in the region of the urethrovaginal septum. Another, is the appearance of secretion at the urethral meatus when the urethra is massaged. A third sign is unusual tenderness of the urethrovaginal area. This last sign accounts for the significant percentage of patients who complain of dyspareunia. All of our patients complained of tenderness in the urethra at one time or another. This seemed to be related primarily to infection, as the tenderness lessened when the infection could be brought under control. Five of our patients had a palpable mass in the roof of the vagina, and secretion could be expressed from the urethra in 7 patients (table 1).

Up to this point we have listed only those diagnostic aids which are within the reach of all physicians. If careful study of the patient in the light of these aids, results in a presumptive diagnosis of urethral diverticulum, urologic help should be sought.

The urologic diagnosis depends upon the discovery of a diverticular opening into the lumen of the urethra or demonstration of the diverticular sac by roentgenography. Examination of the urethra is best done with the McCarthy panendoscope equipped with the foroblique telescope. The orifice of the sac may be extremely small, or large enough to admit the beak of the instrument. It is usually located in the middle third of the urethra on the floor or lateral wall. However, Cook and Pool[2] found 2 cases of diverticula in the files of the Mayo Clinic which opened into the roof of the urethra. Gentle massage of the urethra during the panendoscopic examination usually will cause pus or cellular debris to escape into the irrigating fluid. By tracing this material to it's source the orifice often may be localized.

In many cases, a condé-tipped ureteral catheter may be passed into the diverticulum. If this can be accomplished, filling of the sac with radiopaque material will assist in visualizing it by roentgenography. If a catheter cannot be passed into the diverticulum other methods of filling the sac may be tried. Sometimes simple injection of the urethra with radiopaque media through an olive

FIG. 1. Double diverticula filled with radiopaque medium by means of blunt nosed syringe.

FIG. 2. Double diverticula filled with catheters in each diverticulum. Distortion of diverticulum due to catheters.

FIG. 3. Large single diverticulum filled by the method of Taylor

tipped glass syringe will fill the diverticulum satisfactorily. Moore[3] has credited Taylor[6] with description of a superior technic for demonstrating diverticula, by filling the urethra with radiopaque media under pressure. This is accomplished by tying a silk ligature distal to the bulb of a no. 20 F. Foley catheter and then cutting an orifice immediately proximal to the bulb. The catheter is introduced into the bladder, the bulb is distended and is drawn snugly against the sphincter. The air is forced out of the catheter and the urethral meatus is pinched against the catheter as the urethra is filled with the medium. It is important to empty the diverticulum by massage before attempting to fill it with the radiopaque media.

TREATMENT

The treatment of diverticulum of the urethra is simple to conceive, but due to the fragility of the sac, is sometimes difficult to execute. It generally is agreed that excision of the sac with ligation of it's neck gives the best results. Conservative therapy may be tried in patients with large mouthed small diverticula. This consists of occasional dilation of the urethra by the physician and manual emptying of the diverticulum by the patient following voiding. Small mouthed diverticula, regardless of size, do poorly on conservative therapy. Six of our patients had definitive surgery. One of them was operated upon a second time because she had two diverticula, only one of which was found at the first operation. Five of the patients treated by surgery, are symptom free and the sixth has been operated upon too recently to evaluate. Simple dissection and excision of the sac with ligation or invagination of it's neck was accomplished in all patients

operated upon. Moore's[3] technic of inserting a small Foley catheter through a stab wound into the diverticulum and inflating it's bulb was used in 1 patient. This is a superlative method and aids greatly in the dissection. It is best suited to the larger diverticula. Of our nonoperative patients, 2 are comfortable on conservative therapy and the third prefers her periodic attacks of dysuria to surgery.

SUMMARY AND CONCLUSIONS

Diverticulum of the urethra in the female is a disease which is diagnosed in almost direct proportion to the frequency with which it is considered. Certain characteristics lead to a presumptive diagnosis in most cases.

A definitive diagnosis is an urologic procedure and is made by demonstrating the diverticular orifice in the urethra or by visualizing the sac roentgenographically.

Surgical excision of the sac is usually the treatment of choice.

REFERENCES

1. Carson, R. B., and Wells, W. D.: Treatment of female urethral diverticula, South. M. J. *44:* 285 (April) 1951.
2. Cook, E. N., and Pool, T. L.: Urethral diverticulum in female, J. Urol. *62:* 495 (Oct.) 1949.
3. Moore, T. D.: Diverticulum of female urethra; improved technic of surgical excision, J. Urol. *68:* 611 (Sept.) 1952.
4. Neel, J. C.: Diverticula of female urethra, California State J. Med. *16:* 494 (Nov.) 1918.
5. Priestley, W. O.: Examples of cyst formation in neighborhood of female urethra, Brit. M. J. *1:* 6 (Jan. 2) 1869.
6. Taylor, W. N.: Urologists' Correspondence Club letter *16:* 37, 1950 (quoted by Moore, T.D.[3]).
7. Wharton, L. R., and Kearns, W.: Diverticula of female urethra, J. Urol. *63:* 1063 (June) 1950.

SURGICAL TREATMENT OF PERIPHERAL ARTERIAL DISEASES

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Rapid and extensive changes in the concepts of pathology and treatment of many of the peripheral arterial disorders are occurring at present. Previously accurate diagnosis was difficult in many cases and choice of treatment was limited with the end results frequently unpredictable. Major changes in methods have occurred which provide more material for definitive study of the alterations of structure and function of blood vessels. A better understanding of vascular diseases seems assured.

With the improved technic comes additional responsibility for the physician. Alternatives in treatment compel the making of a correct diagnosis. In the case of diseases of the vascular system where inadequate blood supply often results in extensive and irreversible tissue damage at an alarming speed, the rapidity of such action often is of prime importance. The physician must therefore keep abreast of the newer developments and be prepared to apply them whenever possible.

Classification is as yet inadequate and even the commonly used term, peripheral arterial disease, is somewhat misleading. Some of the disorders have the causative lesion located centrally with the early detectable changes manifested peripherally such as in the so-called Leriche syndrome.

This paper will deal with an over-all survey of these disorders adhering to the standard classification structure. No attempt will be made to go into details of procedure extensively for these are to be found in the articles in the references where excellent descriptions and observations appear.

For a detailed and concise classification of vascular disease the reader is referred to Pratt's text.[9] The diseases of arteries and arterioles are broken into two large groups. 1) Functional (vasomotor) conditions. 2) Organic (structural) conditions. No great change in treatment has occurred in the first group so this discussion will constitute simply a brief review with the emphasis going to the second group wherein several changes have occurred.

I. FUNCTIONAL (VASOMOTOR) CONDITIONS

Raynaud's disease is the best known entity in this classification which includes scleroderma, pernio, acrocyanosis and many others. The exact relation of one to the other is not fully understood. However, they do possess one common characteristic and that is severe vasospasm which is usually not associated with organic obliteration of the vessels in the earlier phases of the disease. The stimulus causing the excessive sympathetic discharge and resulting vasoconstriction of arterioles is widely variable, with cold, emotional stress and tobacco being relatively common causative agents.

These conditions arise predominantly in women, usually during the second to the fifth decades. Most frequently the upper extremities are involved. In the early stage paroxysmal attacks occur which are often manifested by triphasic color changes of pallor, rubor and cyanosis. The subjective symptoms vary according to the type and severity of reaction, for example a burning sensation in the erythromelalgic phase and severe pain during the pallor of the ischemic phase.

Regression usually occurs with advancing age so that conservative management is always the primary treatment. Progression may occur with development of arteriolar fibrosis, thrombosis and necrosis and then surgical intervention may be necessary.

The basis of conservative management is avoidance of precipitating factors whenever possible. Extra protection of the extremities from cold, control of emotion, abstinence from tobacco and even change to a warm climate are valuable measures. Sympathicolytic drugs appear to be of only limited therapeutic value because the resultant systemic vasodilatation shunts the blood elsewhere, reducing the blood pressure and therefore the effective blood flow to the affected extremity. While their use is advocated by some, many others discourage such treatment.

If conservative management is inadequate then sympathetic nerve block with procaine is done to test the vasodilatation in the involved extremity. If the response is satisfactory a surgical removal of the sympathetic ganglia of that extremity is done. Cervicodorsal ganglionectomy with removal of the inferior cervical and the first three or four dorsal sympathetic ganglia is done if the arm is involved. A lumbar sympathectomy with removal of the second to fourth ganglia is done for lower extremity involvement. The measures followed in the conservative management should be continued postoperatively for additional benefit.

II. ORGANIC (STRUCTURAL) CONDITIONS

Thromboangiitis obliterans. Thromboangiitis obliterans is the outstanding example of this group. This disease occurs almost exclusively in men in the third and fourth decades and most often involves the lower extremity. Intermittent claudication in the calf is one of the earliest symptoms, associated with coldness and paresthesia. With elevation of the foot there is pallor and with dependency, rubor. Ulcerations develop with further progression. Rest pain is a later manifestation—probably the result of a neuritis. The peripheral pulses progressively disappear, with the dorsalis pedis and posterior tibial pulses disappearing first, and finally even the femoral pulse. Migratory phlebitis is common. The factors mentioned which aggravate the vasospastic diseases are also harmful in this condition.

Pathologically the process is primarily a panarteritis with thrombosis which attacks mainly the medium sized arteries. There is an associated vasospasm of the involved vessels and even the major collateral arteries. The disease process eventually becomes bilateral. The etiology is unknown but the process is definitely hastened by vasospastic stimuli such as tobacco. An individual or racial predisposition appears important.

Avoidance of any factor causing vasospasm is the first essential in treatment. Care of the extremity to avoid trauma, cold and infection is imperative. Postural exercises supposedly aid in the development of collateral circulation and should be used. Use of the sympathicolytic drugs is controversial for the same reasons as in the vasospastic disorders. Since progression of the disease occurs regardless of conservative measures, bilateral lumbar sympathectomy is indicated early with the above conservative measures being applied postoperatively to improve the prognosis. Prolonged anticoagulant therapy using dicumarol may be of value, particularly if a migratory phlebitis is giving difficulty.[5]

Arteriosclerosis. The changes in concept regarding the proper treatment of this condition have been most striking of late. Previously this very common entity was poorly understood from the pathologic standpoint and results of treatment somewhat unpredictable. The response to lumbar sympathectomy varied in that it would appear to relieve impending gangrene in one case; precipitate it in another; give relief from symptoms in still another, while giving objective evidence of improved circulation in another case and at the same time failing to relieve the intermittent claudication. Current investigations suggest that in order to understand these variations it is necessary to remember that the clinical picture results from combinations of varying pathologic lesions occurring at various anatomic sites. The apparent ability of diabetes millitus to speed up these pathologic changes accounts for their appearance together in a younger age group.

The reason for the development of the arteriosclerotic changes in vessels is not known although many factors are currently highly suspected and in the near future the answer may be forthcoming. Pareira and associates[7] have shown that any distinction between the aging process in elastic arteries and muscular arteries is unrealistic and the usual classification of three separate types of sclerosis, namely atherosclerosis, Monckeberg's medial sclerosis and hyaline intimal proliferation, is not valid. Careful studies of many arteries in the body have led them to conclude that the aging process in arteries starts at an earlier time than was previously appreciated. For example the popliteal artery in a patient aged 20 years already may show significant changes. The aging process may be summarized as follows: First there is intimal thickening due to collagen with an increase in the elastic fibers of the internal elastic lamella and early deposits of calcium along the elastic fibers; later the elastic filaments increase in the intima and media and calcification about them increases; the calcification increases with rock or bone formation; atheromatous plaques appear late in the process and occur in the muscular as well as the elastic arteries. It would appear that the hydrostatic tension in the vessel is an important factor in determining the rapidity of this aging process since its localization and rate correlate with high tension. The incidence of thrombosis at given locations indicates a relation exists between the caliber of the vessel and the degree of atheromatous change.

Such a concept seems to fit well with the observations which emphasize the frequency of a segmental distribution of occlusion in the vessels of the lower extremity. Wylie has reported a series of cases of 57 patients with arteriosclerotic arterial occlusion of which 52 cases were suitable for arteriography with the

occlusion proximal to the popliteal artery. Fourteen of these cases showed extension of thrombosis distal to the popliteal artery while the remaining 38 cases showed a segmental type of thrombosis.[10] Julian, and associates[4] have made similar observations in a considerable number of patients. Wylie and McGuiness[11] have recently reported a series of cases in which stenosis of the vessel has occurred in an isolated segment giving ischemic symptoms.

The anatomic investigations of Edwards,[2] based on dissections and correlation of muscle damage following trauma to specific arteries in the lower extremity, have given a clearer understanding of the clinical pictures encountered. He confirmed the fact that the arterial supply to a muscle is essentially distinct from the other muscles nearby and that there is a lack of significant collaterals so that its circulation is usually a separate unit. A muscle may be supplied by one artery, several branches from one segment of one artery, or occasionally from multiple arteries which are often located in close proximity. Ischemia or necrosis of a muscle or muscle group can therefore occur while the remainder of the limb is unaffected. Clinically this is significant in explaining such problems as claudication in the presence of dorsalis pedis pulsation or failure to relieve claudication by sympathectomy. These observations certainly re-emphasize the multiplicity of factors to be considered in evaluating a given case of arteriosclerosis.

It is unnecessary to review in detail the symptoms and signs of peripheral arteriosclerosis at this time. Instead a few special points will be emphasized. Claudication will vary in its location depending upon which artery is occluded, e.g., in the calf if it is the superficial femoral, in the posterior thigh if the common femoral is involved, in the gluteal and thigh muscles if it is the common iliac, and if there is bilateral common iliac involvement there will be associated impotency. Wylie has emphasized that in stenosis of an artery a combination of a palpable arterial pulse and systolic murmur distal to the stenosis is the most significant finding.[11]

The usual tests of blood pressure, oscillometric readings and skin temperatures should accompany the careful history and physical examination in arriving at the diagnosis. Translumbar aortography or arteriography will be of great use in defining the type and location of the lesion causing the symptoms. This should be done in the cases where extension of the disease process is not so great as to make surgical intervention impossible. It should not be done if the patient is unsuitable because of great age or severe associated disease. The technic described by Wylie and McGuiness is very good.[11] This is usually done under general anesthesia but can be done under local anesthesia with sedation. Arteriography of the peripheral vessels may be done by percutaneous injection or with the vessel exposed. In both procedures it is well to inject 1 cc. of the contrast medium and wait three minutes to test for sensitivity to the agent. Rapid injection and proper timing of the picture are essential for satisfactory information.

The method of treatment will depend on the type and extent of the disease and the general condition of the patient. The routine application of lumbar sympathectomy to all cases of arteriosclerotic ischemia is certainly not indicated.

Freund[3] has reported recently that priscoline caused no appreciable increase in blood flow to muscle as shown by radiosodium studies and he thought that it was of no value in relieving claudication. He believed that intra-arterial injection of the drug into the affected artery might be of some value in maintaining the integrity of the skin in an ischemic extremity. In any event, such agents offer only minimal help, if any, in this problem.

If a segmental type lesion is found to be present, thrombo-endarterectomy is the procedure of choice. The vessel is opened by a longitudinal incision over the site of occlusion. A plane of dissection is developed between the intimal and medial layers with removal of the clot and intima. The vessel is then closed with a continuous suture of no. 00000 arterial silk. Heparinization distal to the point of operation may be used during surgery and for 48 hours postoperatively. The latter may not be necessary, however. Good results in a fairly large series of cases were reported by Wylie who used this technic. Julian and co-workers have also reported satisfactory results with their thrombo-endarterectomy procedure. They also report satisfactory results using the reversed saphenous vein as an autograft to replace the totally resected segment containing the thrombosis. They have used a saphenous vein homograft as well as a quick-frozen preserved aortic bifurcation homograft in 3 patients with success.

Wylie advocates endarterectomy in those patients who are found to have stenosis since this is a prethrombotic lesion and total occlusion is likely to occur. In his series of cases of stenosis, excellent results, anatomic and functional, were obtained in most instances. If the lesion involves the aortic bifurcation or iliac arteries he combines bilateral lumbar sympathectomy with the endarterectomy.

Patients who display multiple areas of segmental occlusion probably represent far advanced disease and are not suitable for endarterectomy. Those having extensive obliteration of the arterial tree are not suitable by the present standards and attempts in this type of case have not met with much success. Lumbar sympathectomy may be done with hope of giving some improvement. However, it is well to remember that presympathectomy tests are not reliable in predicting the benefit to be derived.[8] Sympathectomy may not relieve the claudication and it will at times precipitate or hasten gangrene. Patients for whom even sympathectomy is too rigorous should be treated by postural exercises with alternate elevation, dependency and return to horizontal position several times daily. If gangrene ensues amputation will be necessary, and while there is currently a tendency toward more below the knee amputations because of the greatly lessened disability, the very nature of the disease process will usually make the above the knee amputation the necessary procedure in most cases.

The occurrence of acute thrombosis usually indicates the presence of the previously described arterial changes and endarterectomy or an autogenous vein graft replacement should be done if possible. The general status of the patient is of course a factor in deciding between a conservative management or surgical intervention.

Arterial embolus. Acute arterial embolism involving the extremities occurs usually in the lower extremity from the aortic bifurcation distally. Emboli arise most commonly from the heart in auricular fibrillation or from mural thrombi

secondary to infarction or from the vegetations of endocarditis. The embolus usually lodges at a point of bifurcation where sudden narrowing of the lumen occurs.

The clinical picture is an acute onset of pain and numbness accompanied by pallor, paralysis, loss of arterial pulsation and collapse of the veins.

The sudden occlusion is accompanied by a generalized vasospasm in the extremity. To obtain optimum results the need for therapy is immediate with only a 6 to 10 hour grace period. Aortography and arteriography have been used as an emergency diagnostic procedure with considerable success[6] and should be utilized if aid in diagnosis or location of the embolus is needed. Since some patients will recover without embolectomy, and most of these patients are in the very poor risk category, the following routine as originally outlined by others is probably the best to help separate the surgical from the nonsurgical cases. As soon after onset as possible a procaine sympathetic block should be done. If the extremity warms; has return of color, sensation and function then treatment should be continued using sympathetic blocks as needed or anticoagulant therapy if further blocks are not deemed necessary. If the original sympathetic block is not effective in relieving the picture of ischemia, then surgical exploration of the artery is done at the point of occlusion. A longitudinal incision is made with removal of the embolus; heparin is instilled distally and the vessel is closed with a continuous suture of arterial silk. Even though a few of these limbs might have survived without actual gangrene, severe limitation of function often would ensue. Therefore, this treatment is definitely worth the risk even in the group of patients who admittedly have a poor prognosis because of their primary disease and the possibility of repeated embolic episodes. It is the author's opinion that embolectomy will be used more rather than less in the future. Following embolectomy, anticoagulants should be used for prevention of further embolic episodes.

Trauma. Trauma which involves a blood vessel directly or indirectly may be of such a nature as to require treatment. Here as elsewhere in vascular surgery more definitive and aggressive measures are evolving. The injuries vary from compression by surrounding structures or hematoma, to contusion, perforation or complete division. As a rule there is injury of surrounding structures. However, the vascular injury must have priority of treatment because the time factor is again important. Thrombosis may be occurring at the site of injury or distally, secondary to the vasospasm, and this will often determine the outcome of the case. Cranley and Herrmann[1] emphasize the fact that a blood vessel, while deriving vasoconstrictor impulses from the autonomic nervous system, also has an intrinsic ability to constrict when irritated and will continue this latter type of spasm until the irritating factor is removed regardless of any other therapeutic measures attempted. It is therefore imperative to expose the vessel whenever pulsation is absent so that the irritating factor can be removed and local agents such as papaverine and procaine used to aid in relieving this spasm.

The treatment will of course depend on the type of injury, e.g., simple division may be repaired by suturing; penetrating bone spicules are removed; a compressing extravascular hematoma evacuated; extensive contusion or laceration treated

by resection and ligation or grafting; and a thrombus removed with closure of the vessel. The preferable time interval between injury and treatment is within eight hours. However, exploration is justified up to 24 hours. The surgeon must know which vessels can be ligated with relative safety since that may be a less hazardous procedure and where indicated should be used. The surgeon should of course be fully acquainted with the methods of anastomosing and repairing arteries as well as the sources of grafts if they are necessary.

Postoperatively, shock should be avoided by using whole blood, intra-arterially if necessary, since a lowered pressure can invite thrombosis as well as ischemia. As in all cases of arterial insufficiency the extremity should not be elevated above body level even in the treatment of shock. The choice of additional procedures such as sympathectomy or heparinization will be made by the surgeon depending on the type of injury and operation done. Neither heat nor cold should be applied but rather a temperature of 80 to 92 F. maintained by a simple tent or light wrapping.

Aneurysm. The most commonly encountered aneurysn in the extremities occurs in the popliteal artery. Gangrene is the usual result if this lesion is not treated. There may be cases suitable for excision and replacement by a vessel graft. However, the accepted routine is preliminary lumbar sympathectomy followed in two weeks by aneurysmectomy.

Consideration of all varieties of aneurysm and arteriovenous fistulas of the extremities is beyond the intended scope of this report.

SUMMARY

1. A review of the application of surgical treatment in certain arterial diseases is given.

2. Newer concepts concerning peripheral arteriosclerosis are discussed in terms of their application to the surgical intervention of the disease.

3. Recent changes in the surgical intervention of arteriosclerosis are emphasized.

REFERENCES

1. Cranley, J. J., and Herrmann, L. G.: Definitive treatment of fresh injuries to large blood vessels, A. M. A. Arch. Surg. *67:* 153 (Aug.) 1953.
2. Edwards, E. A.: Anatomic basis for ischemia localized to certain muscles of lower limb, Surg., Gynec. & Obst. *97:* 87 (July) 1953.
3. Freund, J.; Wisham, L. H., and Yalow, R. S.: Effect of priscoline on clearance of radiosodium from muscle and skin of man in normal and diseased limbs, Circulation *8:* 89 (July) 1953.
4. Julian, O. C.; DeTakats, G., and Dye, W. S.: Segmental nature of peripheral arteriosclerosis: surgical application, Angiology *4:* 12 (Feb.) 1953.
5. Linton, R. R.: Treatment of peripheral vascular diseases, Postgrad. Med. *14:* 335 (Oct.) 1953.
6. Lovingood, C. G., and Patton, R.: Translumbar aortography as diagnostic aid in localizing arterial emboli, A. M. A. Arch. Surg. *67:* 164 (Aug.) 1953.
7. Pareira, M. D., Handler, F. P., and Blumenthal, H. T.: Aging processes in arterial and venous systems of lower extremities, Circulation *8:* 36 (July) 1953.
8. Popkin, R. J.: Arteriolar circulation as factor in results obtained in sympathectomy for peripheral arterial occlusive disease, Angiology *4:* 210 (June) 1953.
9. Pratt, G. H.: Surgical Management of Vascular Diseases, Philadelphia, Lea and Febiger, 1949, p. 13.
10. Wylie, E. J.: Thromboendarterectomy for arteriosclerotic thrombosis of major arteries, Surgery *32:* 275 (Aug.) 1952.
11. Wylie, E. J., and McGuinness, J. S.: Recognition and treatment of arteriosclerotic stenosis of major arteries, Surg., Gynec. & Obst. *97:* 425 (Oct.) 1953.

MAJOR SURGERY IN THE NEWBORN

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The prognosis following treatment of congenital anomalies varies considerably. In order to define the contributing factors concerning gastrointestinal anomalies, a review has been made of the abdominal and thoracic operations done during the first 14 days of life for the past 5 years at Colorado General Hospital and at Children's Hospital in Denver. The time interval between birth and diagnosis,[3] the type of lesion, and prematurity are all of importance.

It has been pointed out that the newborn withstand surgery better during the first 24 hours of life than at any time in the next several days.[1] Thus, it is imperative that the physician, under whose care the infant initially comes, be alert to the possibility of serious congenital anomalies. Since 78 per cent of the 69 cases (table I) reviewed here had failure of development of some portion of the gastrointestinal tract which demanded correction, any sign suggestive of obstruction of this system should be viewed with suspicion.

PREOPERATIVE MANAGEMENT

The lesions which bring an infant to the operating room during the first days of life are surgical emergencies. The preoperative program therefore must be rapid as well as effective. Ordinarily, not more than 12 hours should be necessary to bring the patient to optimum condition for surgery.

Since most of these babies cannot be fed by mouth and many are dehydrated, parenteral fluids are necessary. These should be given by vein, usually through a polyethylene cannula placed in an ankle vein.[7] Not more than 40 cc. of fluid per pound of body weight should be given in 24 hours. These infants are easily over-hydrated and one should err on the side of insufficient fluid if there is any question of the quantity to be given. In the premature the volume of saline which can be tolerated is greatly reduced because of immature kidneys. Ordinarily no salt is necessary. If this electrolyte must be given, it should be administered in the form of 0.45 per cent saline and never in amounts greater than 20 cc. per pound. To give more invites the development of pulmonary edema.

In the newborn and especially in the premature, plasma protein may be low and a reduced erythrocyte count and hemoglobin level frequent. The infant tolerates poorly a change in blood volume.[4] If the hemoglobin level is below 10 grams per 100 cc. a transfusion of whole blood is given. Blood should, of course, be available for use in the operating room.

The infant is particularly susceptible to distention of the gastrointestinal tract and efforts to prevent this are of value. Gastroduodenal suction using a no.

TABLE I

Primary diagnosis

1. Tracheo-esophageal fistula... 38
2. Atresia and stenosis of intestinal tract............................... 9
3. Imperforate anus.. 7
4. Omphalocoele... 7
5. Incomplete rotation of the colon with duodenal obstruction.......... 4
6. Diaphragmatic hernia... 4

      Total... 69

8F urethral catheter which has been sterilized enough times to insure its softness is satisfactory. By keeping the bowel deflated in such fashion, respiration is improved and operative manipulations within the abdomen facilitated.

Pulmonary complications form a real problem both preoperatively and postoperatively. This is particularly true in the infant with esophageal atresia.[5] Aspiration of oropharyngeal secretions should be minimized by removal of these secretions with a soft rubber catheter attached to a mechanical suction apparatus. Antibiotics in the form of penicillin and streptomycin are important in the prevention of pneumonia and the protection of the operative site from infection.

There has been ample demonstration by laboratory studies that Vitamins C and K are of great importance in the neonatal period. In the premature especially, a severe hemorrhagic tendency may develop by the third day of life due to hypoprothrombinemia.[4, 8] This should be anticipated with daily injection of Vitamin K. Administration of Vitamin C is of value in correcting the incomplete metabolism of amino acids and promoting sound wound healing.[6]

An environment of constant temperature, adequate oxygen concentration and proper humidity is important in maintaining life in this uncertain period. This is best insured by use of an incubator. The premature infant withstands handling and manipulation poorly and the less he is disturbed the more rapidly he progresses.

OPERATIVE CARE

It is beyond the scope of this paper to discuss the technical aspects of correction of the various anomalies encountered (table II). There are, however, a few points common to all surgery in the neonatal period. The importance of protecting the blood volume by transfusion and the prevention of over-hydration have been discussed. Maintenance of body heat is a problem which needs attention. Temperature changes are poorly tolerated and the use of a hot water bottle under the baby and wrapping of the extremities are of value.

Operative speed has been emphasized in the literature as essential when dealing with newborns. We were unable to correlate duration of operation with ultimate outcome in this series and believe that gentleness, asepsis and hemostasis are much more important than a brief anesthetic. Although the premature withstands hypoxia somewhat better than the older child, every effort should be made to maintain adequate oxygenation during anesthesia.

TABLE II

Types of congenital anomalies found

1. Tracheo-esophageal fistula...38
2. Atresia and stenosis of intestinal tract.............................9
3. Imperforate anus..8
4. Omphalocoele..7
5. Diaphragmatic hernia..4
6. Patent ductus arteriosis..3
7. Incomplete rotation of colon with duodenal obstruction.............3
8. Interventricular septal defects.....................................3
9. Extrophy of bladder...2
10. Horseshoe kidney...1
11. Incomplete attachment of mesentery................................1
12. Extrophy of colon..1
13. Atresia of tricuspid valve...1
14. Absence of radius..1
15. Meconium ileus with fibrocystic disease of the pancreas...........1
16. Atresia of extra hepatic bile ducts...............................1
17. Cystic disease of kidney...1
18. Meckel's diverticulum..1
19. Bronchial cleft cyst...1
20. Mongolism..1
21. Harelip..1
22. Dextra position of aorta...1
23. Absence of kidney..1
24. Hypoplasia of kidney...1

Total...92

POSTOPERATIVE PROGRAM

The principles discussed in the preoperative management apply following operation. Particular attention should be paid to the baby's protein status and if oral feeding cannot be started within a few days of surgery, protein in the form of plasma or salt poor human albumen should be given by vein. The hemoglobin level must be followed and adequately maintained.

When oral feedings can be started, the use of a gavage tube is frequently necessary. The premature usually, and the term newborn often, are not strong enough to nurse from a nipple and formula should be given by gastric tube. The change to bottle feeding is dictated by the speed with which the infant progresses.

Brief mention should be made of the importance of abdominal dressings which are loose and nonconstricting. In the normal adult vital capacity is reduced 30 per cent simply by application of an abdominal binder.[2] Since respiration in the newborn is largely diaphragmatic, tight bandages over the upper abdomen can seriously inhibit oxygenation.

ANALYSIS OF CASES

In this series 25 premature infants were operated upon; 18 of these died—a mortality rate of 72 per cent. There were 42 full term babies of whom 13 died—

a mortality rate of 30.9 per cent. Two additional infants died whose weights were unknown. The 2 groups may be compared as each contained, in approximately equal numbers, the various types of congenital anomalies listed.

Twenty-five of the 69 patients had multiple anomalies. Twenty-four different congenital lesions were encountered in the group, of which 9 were uncorrected at operation. All of these 9 were incompatible with life and these infants died either as a direct result of the anomalies or from complications arising from them. Multiple lesions incompatible with life were encountered in premature infants nearly four times as frequently as in full term infants.

Esophageal atresia with tracheo-esophageal fistula was the lesion most frequently found. When this was associated with an intra-abdominal stenosis or atresia of the intestinal tract the outcome was invariably fatal. When the two types of lesion are associated the diagnosis is not only difficult but adequate treatment requires multiple operations.

Dehiscence of the surgical anastomoses in infants with esophageal atresia caused the most fatalities—17 of the 33 deaths (table III). Invariably this was

TABLE III

Causes of death

1. Tracheo-esophageal fistula—dehiscence of surgical repair	17
2. Peritonitis—generalized	5
3. Pneumonia	4
4. Multiple uncorrected anomalies of intestinal tract	3
5. Congenital cardiac anomaly with cardiac failure	2
6. Cardiac arrest during operation	1
7. Spontaneous pneumothorax	1
Total	33

the result of too much tension on the suture line. A short proximal segment or a long area of hypoplasia in the distal segment usually was the cause of this.

Generalized peritonitis was the second most common cause of death. This resulted either from multiple uncorrected atresias in the bowel with perforation or in 1 case from a leak in the suture line.

Pneumonia was responsible for four deaths although there were contributing factors; pulmonary edema and sepsis were the most prominent. Faulty electrolyte balance was contributory in one instance.

DISCUSSION

The problem of emergency surgery in the first 14 days of life is first of all that of the repair of congenital anomalies. Specifically, esophageal atresia with tracheo-esophageal fistula is the most difficult to treat from a purely technical standpoint. Too much tension on the suture line with dehiscence of the anastomosis is the most common single cause of death in this group of cases.

Prematurity is the factor which contributed to more deaths than any other. Seventy-two per cent of the premature infants in this series died. This group

should not be confused with other series of premature infants surviving after operation which includes the repair of pyloric stenosis.

Finally, the cause of death in 9 instances was directly or indirectly attributable to multiple uncorrected anomalies incompatible with life.

SUMMARY

Sixty-nine infants were operated upon for one or more congenital anomalies in the first 14 days of life. There were 33 fatalities or 48 per cent.

Twenty-four different congenital anomalies were encountered; tracheoesophageal fistula was the most common.

Dehiscence of the suture line following esophageal anastomosis for atresia was the most common cause of death.

Prematurity was the most common contributory cause of death.

In 9 instances death was directly attributable to multiple uncorrected anomalies incompatible with life.

The preoperative and postoperative care of the newborn is discussed.

REFERENCES

1. Arnheim, E. E.: Surgery of newborn, J. Mt. Sinai Hosp. *17:* 528 (March–April) 1951.
2. Churchill, E. D., and McNeil, D.: Reduction in vital capacity following operation, Surg., Gynec. & Obst. *44:* 483 (April pt. 1) 1927.
3. Fisher, H. C.: Duplications stenosis and atresia in infancy, Am. Surgeon *18:* 952 (Oct.) 1952.
4. Gross, R. E., and Ferguson, C. C.: Surgery in premature babies; observation from one hundred and fifty-nine cases, Surg., Gynec. & Obst. *95:* 631 (Nov.) 1952.
5. Grow, J. B., and Neerken, A. J.: Esophageal atresia and tracheoesophageal fistula, J.A.M.A. *152:* 1614 (Aug. 22) 1953.
6. Levine, S. Z.; Gordon, H. H., and Marples, E.: Defect in metabolism of tyrosine and phenylalanine in premature infants; spontaneous occurrence and eradication by vitamin C, J. Clin. Investigation *20:* 209 (March) 1941.
7. Potts, W. J.: Minor surgical problems of newborn, S. Clin. North America *31:* 1441 (Oct.) 1951.
8. Robarts, F. H.: Surgical emergencies of infancy, Edinburgh M. J. *59:* 409 (Sept.) 1952.

SURGICAL CLOSURE OF ATRIAL SEPTAL DEFECT: THE RESPONSE IN A PATIENT WITH SEVERE PULMONARY HYPERTENSION

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Atrial septal defect is one of the common congenital malformations of the heart. The lesion usually occurs in two locations depending upon the nature of the embryologic arrest in the formation of the septum. The most frequent location is in the area of the foramen ovale, thus a central location; less commonly the defect lies low in the septal wall in close proximity to the atrioventricular valves. Occasionally there are multiple openings; and in addition, frequently are associated anomalies of the pulmonary venous return. When the septal defect occurs low, immediately above the atrioventricular valves, the technical difficulties of closure are greater than when the defect occurs higher in the septum. Regardless of location the defect gives rise to a left-to-right shunt of blood which results in an elevated pulmonary blood flow. Depending upon the magnitude of this pulmonary blood flow varying degrees of enlargement of the right atrium, right ventricle and pulmonary vasculature result. Correspondingly the natural history of patients with this defect shows wide variation in symptomatology and in their life span.

It is recognized that many patients with smaller defects and lesser magnitudes of pulmonary blood flow may lead relatively normal lives and live to middle age or beyond. Large defects may result in pulmonary flows of such a magnitude that congestive failure develops early in life. In other instances the development of pulmonary hypertension is the limiting factor as regards the natural history of patients with this defect.

The factors leading to the development of pulmonary hypertension are not clearly understood, but may include the volume of pulmonary flow and the development of obliterative pulmonary vascular changes.

Therefore, since this lesion is common and frequently results in disability and premature death it constitutes an important challenge for the surgeon to develop safe and adequate technics for its closure.

SURGICAL BACKGROUND

The experimental approach to the solution of the problem of adequate closure of atrial septal defects was initiated by Cohn[3] in 1947. Since that time many

ingenious but indirect technics have been evolved to accomplish this purpose. These recently have been reviewed by Bailey and associates[1] and by Swan.[5]

Experience in this institution with several types of indirect technics for the closure of atrial defects led to the realization that, despite good results in the experimental laboratory, when applied to very large defects found in the clinical patient, indirect methods did not afford complete closure of the defect. Accordingly, we have recently turned to the method first described by Lewis and Taufic[4] of direct vision closure with the aid of hypothermia and cardiac inflow occlusion. The technical aspects of this procedure have recently been reported by Swan and associates.[6]

Briefly, the body temperature of the anesthetized patient is lowered to approximately 30 C. in a tub of ice water. The patient is then removed to the operating room with a continued fall in body temperature to the neighborhood of 24 C. An anterior transverse sternum splitting bilateral thoracotomy is then done. At this temperature it is considered safe to totally occlude circulation for periods up to 10 minutes. The right auricle is then opened and the septal defect closed under direct vision with interrupted silk sutures. It is of the utmost importance that measures be taken to prevent coronary air embolism at the time of closure of the right atrium.

CASE REPORT

G. H., a 26 year old white woman had experienced ease of fatigue and exertional dyspnea since early childhood. A cardiac murmur was first detected at 5 years of age and bed rest was instituted for one year. Dyspnea and fatigue progressed until at the age of 15 years the exercise tolerance was limited to three blocks. At age 20 years a diagnosis of congenital heart disease was made and the patient was advised to avoid exertion and pregnancy. At age 26 years the patient was unable to walk one block without discomfort, and was forced to sleep with three pillows because of shortness of breath.

The physical examination revealed enlargement of the heart to the left anterior axillary line. There was an over active precordium with a forceful cardiac impulse palpable in the left third and fourth intercostal spaces between the midsternal and midclavicular lines. A systolic thrust followed by a shock was palpable in the left second intercostal space. Auscultation revealed a marked increase in the intensity of the second heart sound in the left second interspace with fine reduplication. A grade III systolic murmur was present along the left sternal border with maximum intensity in the left third intercostal space. This murmur was high pitched and rough in quality and was transmitted toward the apex. The blood pressure in the arm was 105/75 mm. Hg. The lungs were clear and the liver was not enlarged. There was no cyanosis or clubbing, and the femoral arterial pulsations were normal.

The electrocardiogram (fig. 1) demonstrated an r s R' pattern in precordial position V-1. The R' wave was 18 mm. in amplitude and showed a delayed intrinsicoid deflection time of .05 seconds. The total QRS duration was .10 seconds. There were prominent upright P waves in the right sided precordial leads. The tracing was interpreted as indicating right ventricular hypertrophy with incomplete right bundle branch block and probable right atrial enlargement.

The fluoroscopic examination showed a considerable increase in the vascularity of the lung fields. The main pulmonary artery and the left and right pulmonary arteries were greatly increased in size and showed significant increase in the amplitude of pulsations. The right atrium was greatly enlarged. The ventricular area was considerably enlarged with the configuration suggesting right ventricular enlargement (fig. 2).
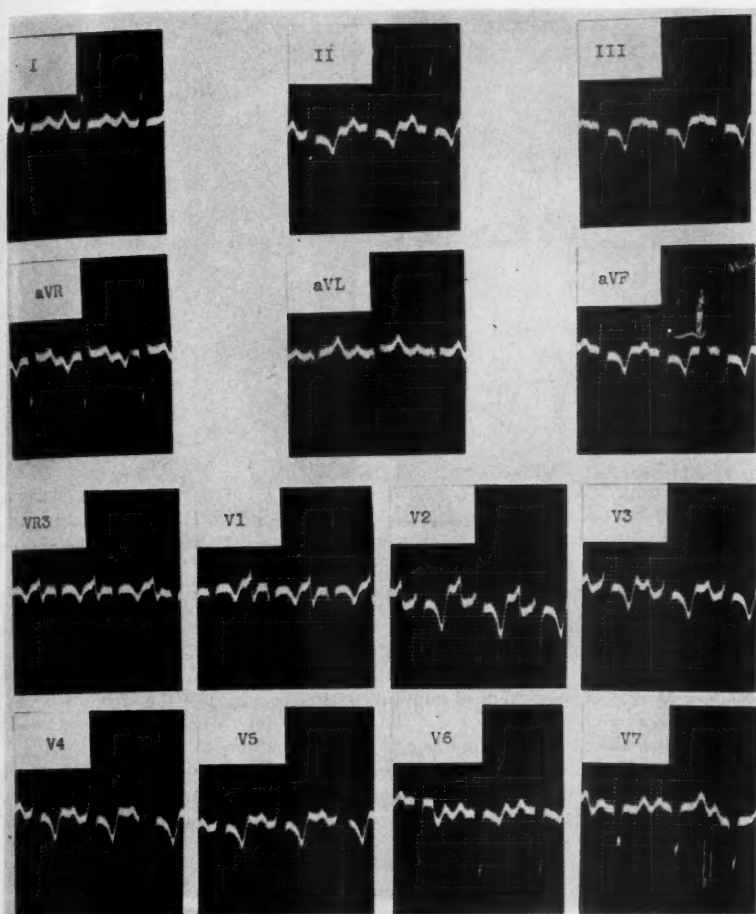
Fig. 1. The electrocardiogram demonstrates a pattern of right ventricular hypertrophy with incomplete right bundle branch block, and right atrial enlargement.

Cardiac catheterization was done via a left basilic vein on March 5, 1953. The catheter pursued an abnormal course indicating the presence of a persistent left superior vena cava entering the right atrium through the coronary sinus. The catheter was advanced into the right ventricle and into the left atrium and left ventricle. Blood samples indicated no anomalous pulmonary venous drainage into this persistent left superior vena cava. A second cardiac catheterization was done on April 14, using the right basilic venous system and the results are shown in table I. The pulmonary artery was intubated without difficulty and the catheter was again passed into the left atrium and thence into a pulmonary vein. The study indicated the presence of a left to right shunt at the atrial level with a pulmonary flow of 8.9 liters/min./M^2. There was a marked elevation of the pulmonary arterial pressure to average levels of 100/30 mm. Hg (table I).

On April 15 a thoracotomy was done using a transternal approach under hypothermia at
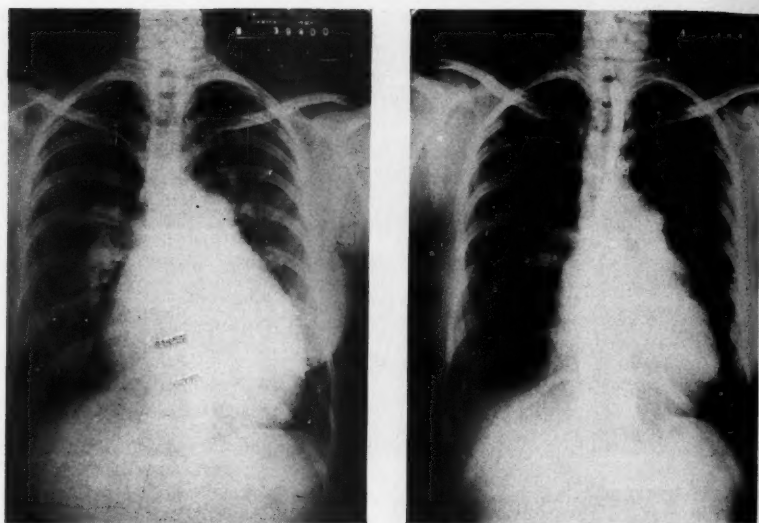
Fig. 2. The preoperative chest roentgenogram is shown on the left and four months post-operative on the right. Decrease in heart size and decreased vascularity of the lung fields is apparent.

21.5 C. Marked enlargement of the pulmonary artery and right atrium was apparent on exposure of the heart. The vena cavae were occluded, the lateral wall of the right atrium was clamped and an incision was made. The right atrium was opened, and a 2.5 by 4.0 cm. atrial defect was closed and circulation re-established after occlusion of the circulation for 7.5 minutes. Following closure of the chest wall and warming to normal body temperature shock presented which responded to blood transfusion and pressor agents.

Postoperatively a rapidly changing succession of supraventricular cardiac rhythms occurred. The postoperative course was further complicated by a transient partial aphasia which disappeared within 48 hours. The patient was discharged on May 18, 1953.

TABLE I

Preoperative physiologic studies

Catheter Position	Pressure Systolic/ Diastolic mm. Hg	Oxygen Content cc./liter	Oxygen Saturation Per cent
Superior vena cava................	—	104.0	54.9
Inferior vena cava...............	—	124.8	65.9
Right atrium*....................	11/5	161.3	85.1
Right ventricle*.................	100/8	164.1	86.5
Pulmonary artery*...............	100/30	163.8	86.4
Left atrium.....................	14/5	177.1	93.4
Pulmonary vein..................	15/4	178.6	94.3
Pulmonary capillary.............	12 (mean)	—	—
Brachial artery.................	113/84	179.5	94.7

Oxygen consumption: 203 cc./min. Systemic blood flow: 2.3 l./min./M². Pulmonary blood flow: 8.9 l./min./M².

* Average of two blood samples.

TABLE II

Postoperative physiologic studies

Catheter Position	Pressure Systolic/ Diastolic mm. Hg	Oxygen Content cc./liter	Oxygen Saturation Per cent
Superior vena cava................	—	103.9	59.1
Right atrium*.....................	2/1	122.3	69.5
Right ventricle*..................	30/0	124.8	71.0
Pulmonary artery*................	30/17	124.6	69.8
Pulmonary capillary*.............	9 (mean)	—	—
Brachial artery	104/63	168.9	96.0

Oxygen consumption: 204 cc./min. Systemic and pulmonary blood flow: 3.0 l./min./M².
* Average of two blood samples.

Physical activities were progressively increased so that within four months the patient could walk a distance of half a mile without discomfort and was able to attend and participate in dances. The patient has been able to sleep comfortably with one pillow. There has been a 12 pound weight gain in the four months since discharge from the hospital.

Physical examination at four months revealed definite decrease in the heart size with no palpable thrusts or shocks. On auscultation the second heart sound in the left second interspace was reduplicated and only slightly increased in intensity. No murmurs were present along the left sternal border. A grade I systolic murmur was audible at the apex. The electrocardiogram demonstrated atrial flutter with a varying ventricular response.

Fluoroscopic examination revealed a decrease in the vascularity of the lung fields. A marked decrease in the amplitude of pulsations in the pulmonary arteries was noted as compared to those present preoperatively. The right atrium and right ventricle remained enlarged but showed a striking decrease in the size since surgery (fig. 2).

Cardiac catheterization done on Sept. 10, 1953 (table II), revealed a normal oxygen saturation of the mixed venous blood at the pulmonary arterial level of 69.8 per cent indicating an absence of a left to right shunt. There was a dramatic decrease in the pulmonary arterial pressure to average levels of 30/17 mm. Hg.

DISCUSSION

The patient discussed in this paper was a 26 year old woman with a large atrial septal defect and pulmonary hypertension. The physiologic data presented revealed that the defect was completely closed at the time of operation. Thus, this case demonstrated the feasibility of complete closure under direct vision of large atrial septal defects despite the very considerable enlargement of the heart, the age of the patient, and the significant pulmonary hypertension.

Now that it has been established that such defects can successfully be closed it is of the utmost importance that accurate clinical evaluation of these patients be pursued. The clinical diagnosis of atrial septal defect has been discussed in the past by several authors.[1, 2] Once the diagnosis has been established the problem of the selection of suitable candidates for surgical correction arises. The great variability in the natural history of patients with this defect is well known. Thus, at this stage in the development of the operative procedure it would be of great help could one predict those patients who will enjoy relative longevity as compared to those who will develop complications leading to serious disability early in their lives. However, a review of the studies of our patients with atrial

septal defects reveals that such a prediction is difficult, if not impossible to accomplish.

Early in the history of any operative technic it is common to wait until the patient is actually a very poor risk for the operative procedure before advising surgical intervention. The indications for operation are changed as experience grows until finally patients with relatively few or early symptoms are suggested for operation. If the operative risk can be demonstrated to be sufficiently low with this procedure this well may be the case as regards the patient with an atrial septal defect. At the present time we have successfully closed atrial septal defects by the method described in 4 patients with death in a fifth. It is considered by the authors that, in the future, atrial septal defects may be closed merely because of the presence of such a defect regardless of the clinical status of the patient. The criterion for operation will thus be similar to that in patients with a patent ductus arteriosus, namely the diagnosis of the anomaly.

CONCLUSIONS

The clinical course, the operative technic, and physiologic studies of a 26 year old woman with an atrial septal defect are presented.

The postoperative course, the physical and fluoroscopic findings and the physiologic data all indicate complete closure of the defect.

This patient demonstrates that severe pulmonary artery hypertension does not constitute a contraindication to closure of an atrial septal defect provided an increased pulmonary blood flow is present.

REFERENCES

1. Bailey, C. P., and others: Congenital interatrial communications: clinical and surgical considerations with description of new surgical technic: atrio-septo-pexy, Ann. Int. Med. 37: 888 (Nov.) 1952.
2. Barber, J. M., Magidson, O., and Wood, P.: Atrial septal defect with special reference to electrocardiogram, pulmonary artery pressure and second heart sound, Brit. Heart J. 12: 277 (July) 1950.
3. Cohn, R.: Experimental method for closure of interauricular septal defects in dogs, Am. Heart J. 33: 453 (April) 1947.
4. Lewis, F. J., and Taufic, M.: Closure of atrial septal defects with aid of hypothermia; experimental accomplishments and report of one successful case, Surgery 33: 52 (Jan.) 1953.
5. Swan, H.: Surgical closure of interauricular septal defects, J.A.M.A. 151: 792 (March 7) 1953.
6. Swan, H., Zeavin, I., Blount, S. G., Jr., and Virtue, R. W.: Visual surgery in open heart during hypothermia, J.A.M.A., to be published.

RECENT TRENDS IN ANESTHESIOLOGY

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ELIMINATION OF CARBON DIOXIDE

The most important widespread trend in anesthesiology today is due to the realization that breathing is essential as much or perhaps even more in order to remove carbon dioxide from the body as to get oxygen into the body. This was stressed several years ago by Moyer and Beecher,[20] who found that ventilation adequate to give good oxygenation failed to remove carbon dioxide from the patients. At about the same time, Stormont and associates[26] reported that when tidal volume is reduced, respiratory acidosis occurs with a corresponding increase of plasma pH in cyclopropane anesthesia. Further proof that this is true was given in the diffusion respiration experiments of the past few years made by Whitehead and Draper,[9] who found that oxygen is taken into the body under conditions when the lungs do not move at all, whereas carbon dioxide is not removed from the body under those same conditions. It is known that an excess of carbon dioxide depresses conduction in the heart severely enough so that a heart block may occur when the pH is lowered to 7.0[2] The sinuauricular node becomes depressed, and the heart beat is slow and irregular. Circulatory failure is most likely due to heart failure rather than the failure of the vasomotor center.

Many different workers have recently stressed the dangers of allowing carbon dioxide to accumulate. Gabbard[12] states that considerable ventilation is required to prevent carbon dioxide accumulation but that adequate ventilation will prevent respiratory acidosis. In order to be adequate however, ventilation must be far beyond what the anesthesiologist would consider to be average respiration when the chest is not opened. Arrhythmias, lowered blood pressure, convulsions, apnea and cardiac arrest, according to Gabbard, may be the direct result of accumulation of carbon dioxide. Gray and Rees[13] stress the fact that adequate ventilation eliminates carbon dioxide and that an excess of ventilation will inhibit the Hering-Breuer reflex which diminishes respiratory center activity; that in turn results in lack of motor neuron activity which is followed by a lowered tonus of respiratory muscles so that less curariform agent is required for relaxation of the patient. They therefore believe that controlled respiration takes a considerable load from the patient.

Maier and his co-workers[18] state that adequate information concerning acidosis during operation cannot be obtained by determinations made only at the beginning and the end of operation. They believe that continuous recordings of the pH would be preferable and think that a quiet field in thoracic surgery may be obtained at the expense of acidosis to the patient. Bunker and others[4] observed that acidosis occurs during cyclopropane anesthesia and Beecher[1] gave figures to

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indicate that it is very difficult to get rid of carbon dioxide during thoracic surgery. Taylor and Roos[28] emphasized that respiration must be continually aided in order to properly eliminate carbon dioxide. Johnstone[15] found that building up of carbon dioxide during cyclopropane anesthesia was followed by many arrhythmias and suggested that most of the so-called cyclopropane arrhythmias are due to the presence of carbon dioxide. Hyperventilation in his cases removed the carbon dioxide and was also followed by disappearance of the arrhythmias involved.

Orton[23] states that it is far safer to overventilate patients than it is to underventilate them, and that special care is necessary to insure good ventilation in patients receiving hypotensive drugs. In the absence of good ventilation, any capillary dilatation due to carbon dioxide will be imposed upon the arteriolar dilatation produced by the hypotensive drug and grave trouble will arise quickly. A word of warning comes from some work of Miller[19] and associates who gave animals high concentrations of carbon dioxide for about two hours and then suddenly hyperventilated the animals with air or oxygen. More than half the animals soon went into ventricular fibrillation. Although the reasons for this are immediately not apparent, it would seem that it might be dangerous to allow carbon dioxide to build up in patients and then to suddenly hyperventilate them. Adequate breathing for the patients from the beginning of the anesthetic period would seem to be a much better and safer procedure.

Young[31] and co-workers have shown that in dogs carbon dioxide considerably prolongs the time before vagal escape occurs after the dogs have had their vagus nerves electrically shocked. That work has been repeated and confirmed in this laboratory.[25] These results indicate that accumulation of carbon dioxide may be very potent in accentuating the so-called vago-vagal reactions which may result in cardiac arrest. Since agents which cause relaxation of the abdominal muscles also cause relaxation of thoracic muscles, it is wise to assist ventilation of any patient to whom relaxing agents have been given in order to adequately remove carbon dioxide.

TRICHLOROETHYLENE

An agent which seems to have taken a definite place in anesthesiology in the past few years is trilene or trichloroethylene. It is a good analgesic agent but is not a potent anesthetic agent; that is, it gives relief from pain but is not efficacious in producing sleep. It is valuable in that it may be used for analgesia; in fact, the patient can give the anesthetic to himself. If an inhaler is fastened to the patient's wrist, the patient can use the inhaler so long as analgesia is necessary. If the patient should fall asleep, the inhaler will fall away from the face to be used again when the patient desires. Trichloroethylene is a sparingly volatile liquid which smells like chloroform but is much less potent. Signs of over-dosage are easily apparent because the patient hyperventilates considerably with too much drug. This agent does not give relaxation and should not be used when relaxation is desired. Tremors frequently occur. Early work with dogs indicated that when epinephrine is injected into animals getting trichloroethylene anes-

thesia, arrhythmias frequently occurred.[29] On that basis, its use in this country was delayed for some years; however, it is being used now with comparative safety.

A much smaller ratio of deaths with trichloroethylene than with other agents has been reported.[17] Trichloroethylene is a valuable agent for such procedures as myringotomies, for incision and drainage, for reduction of a fracture, or for cystoscopic examinations in poor-risk patients. Trichloroethylene is decomposed by soda lime forming toxic products so that it should not be used in the presence of soda lime. This practically precludes its use in a closed system. It is used either with an inhaler so that air is drawn through the inhaler vaporizing the trichloroethylene or it can be used with nitrous oxide so that a mixture of nitrous oxide and oxygen blows over the trichloroethylene thus vaporizing the agent. Recovery after the use of trichloroethylene is usually very rapid. However, in a few instances it has been prolonged, and when more than necessary was given recovery has taken as long as 24 hours.

RELAXING AGENTS

Much development in the field of relaxing agents has occurred recently. A good deal of work has been done on the mechanism of their action. It has been fairly well agreed that curare, dimethyltubocurarine and flaxedil act by preventing acetylcholine from depolarizing the motor end-plate. Another group of relaxing agents, namely decamethonium (syncurine) and succinylcholine, act to prevent repolarization of the motor end-plate.[11] It is seen therefore, that the mechanisms of the actions of these two groups of agents are opposed to each other and that if they are used in rapid succession on the same patient the use of the second rather than causing relaxation may increase muscle tonus. The two types of compounds act antagonistically.

Preparations of d-tubocurarine were the earliest relaxing drugs to be used. A dose would last perhaps 20 minutes. The dimethyl compound is more potent and its action may be expected to last 30 minutes. Flaxedil is a purely synthetic substance the action of which may be expected to last 15 minutes. Flaxedil, in addition to causing muscle relaxation, has a definite vagolytic effect so that a fourth to a third of the patients receiving it will be noted to have an increase in pulse rate. Decamethonium acts for perhaps 10 to 15 minutes, depending, of course, upon the dose. This type of drug, because it does depolarize the end-plate, may be expected to cause muscle contraction and in fact, it does. Fasciculations are the rule rather than the exception when syncurine or succinylcholine is administered intravenously. The definite advantage afforded by succinylcholine is that it is usually metabolized within two to three minutes so its action is relatively short. Consequently the patient is safer than if he had had a drug which relaxed him and diminished his respiration for a longer period of time. Succinylcholine may be used for intubations when relaxation for the operation is not needed.

A few instances have been reported of prolonged action of succinylcholine. These reports seem to be due to the fact that some patients have a lower pseudocholinesterase in their plasma than normal. Normal patients are known to have

true cholinesterase in their red blood cells which has the property of hydrolizing acetylcholine, but which will not hydrolize succinylcholine. Pseudo-cholinesterase exists in the plasma. It hydrolizes succinylcholine, thus preventing succinylcholine from tying up the true cholinesterase. In patients who have a low pseudo-cholinesterase, the succinylcholine ties up all of the pseudo-cholinesterase and true cholinesterase and prevents the true cholinesterase from acting until enough pseudo-cholinesterase becomes available to hydrolize the succinylcholine.[10] It is hoped that a commercial preparation which antagonizes succinylcholine soon will be available.

Because of the excess salivation and bronchorrhea, mytolon, another relaxing agent, has not come into very widespread use. Much work is continuing on the preparation of new relaxing agents.

<center>LOCAL ANESTHETICS</center>

For many years procaine has been the local anesthetic agent of choice. For longer procedures such drugs as nupercaine and pontocaine have had their proponents. Many other local anesthetic agents have been made in attempts to find compounds which were either more potent, longer acting, or less toxic than procaine, but they have not been very successful. In the past few years, xylocaine (lidocaine) seems to have made a place for itself because of properties which are favorable in some circumstances. It is about twice as potent as procaine without being any more toxic. Its solutions seem to spread better than do solutions of procaine, especially epidurally. Epidural blocks with procaine have resulted in spotty analgesia while those with xylocaine seem to be very satisfactory.[7] Xylocaine has the advantage of being effective topically and is available in ointments. Solutions of xylocaine with epinephrine are stable for considerable periods of time. This is not true of solutions of procaine with epinephrine. Xylocaine solutions can be boiled without losing any appreciable potency.

About two years ago a new vehicle was introduced to keep procaine from rapidly being absorbed. The new combination of substances was termed efocaine. Many reports of its valuable use appeared and it seems that when it is put on a nerve trunk, anesthesia may be expected for several days up to periods lasting as long as two weeks. A few cases of neuritis have occurred after its use. The manufacturers state that it should not be used where it can be allowed to pool. When used epidurally, its action lasts only about 24 hours. While this agent seems to be of some value, it cannot be accepted without reservations to date.

<center>HYPOTENSION</center>

Many operations have been done using hypotensive anesthesia. One advantage suggested for this method is the saving of blood because patients whose pressures are quite low do not bleed so readily. This enables the surgeon to have a relatively bloodless field. The patients who recover from hypotensive anesthesia seem to become alert more rapidly than those who have had larger quantities of anesthetic drugs. On the other hand, Little and associates[14] have examined the

complications following hypotensive anesthesia in a series of over 21,000 cases. They found that one death in 459 cases—approximately .2 per cent—due to the anesthetic agent was reported. This is a much higher percentage than the number of deaths reported by other methods and with other agents. It would seem to this author that hypotensive anesthesia should be reserved for those situations in which it is questionable as to whether blood could be obtained for a patient to keep his blood pressure near normal. There may be a few definite indications for this type of anesthesia, but the death rate from the method itself is high enough so that it should not be used unless the anesthesiologist thinks that the risks in using this method are definitely outweighed by the risk of not using it.

REFRIGERATION ANESTHESIA

For several years attempts have been made to devise methods whereby cardiac surgery can be done under direct visualization. Many devices have been made in attempts to short-circuit the lungs and heart while the surgeon works on the heart. These are constantly being improved, but they are not easily available to everyone and require a great deal of attention.[30] Reports have appeared[3, 16] stating that hypothermia diminishes metabolism of the brain sufficiently so that circulation to the heart could be cut off for as much as 10 minutes while direct surgery on the heart was being done. This method has been used in several centers in this country.[3, 16, 27] Although it is relatively simple, there is danger that ventricular fibrillation will occur in some patients and the instances when this may occur are unpredictable. There is evidence that the patient should be hyperventilated during hypothermia. Recovery from this procedure seems to be very satisfactory, probably because very little in the way of anesthetic agent is given. It is necessary to put the patient to sleep before he is cooled. Several successful procedures in which hypothermia has been used include direct closure of intra-atrial septal defects and visual attack on pulmonary stenoses.[16, 27]

The hypotension obtained by the use of hypothermia has been used successfully several times when complete occlusion of the circulation was not desired. This type of hypotension may be better tolerated by the patient than that obtained by the use of drugs.[5, 21]

A preliminary report[8] indicates that further developments in this field are forthcoming.

REFERENCES

1. Beecher, H. K., and Murphy, A. J.: Acidosis during thoracic surgery, J. Thoracic Surg. 19: 50 (Jan.) 1950.
2. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 5, Baltimore, Williams & Wilkins Company, 1950.
3. Bigelow, W. G., Callaghan, J. C., and Hopps, J. A.: General hypothermia for experimental intracardiac surgery, Ann. Surg. 132: 531 (Sept.) 1950.
4. Bunker, J. P., and others: Metabolic effects of anesthesia; comparison of acid-base equilibrium in man and in dogs during ether and during cyclopropane anesthesia, J. Pharmacol. & Exper. Therap. 102: 62 (May) 1951.
5. Collins, V. J.: Personal communication to the author.
6. Cookson, B. A., Neptune, W. B., and Bailey, C. P.: Hypothermia as means of performing intracardiac surgery under direct vision, Dis. Chest 22: 245 (Sept.) 1952.

7. Crawford, O. B.: Peridural anesthesia for thoracic surgery, New York State J. Med. 52: 2637 (Nov. 1) 1952.
8. Delorme, E. J.: Experimental cooling of blood stream, Lancet 2: 914 (Nov. 8) 1952.
9. Draper, W. B.; Whitehead, R. W., and Spencer, J. N.: Studies on diffusion respiration; alveolar gases and venous blood pH of dogs during diffusion respiration, Anesthesiology 8: 524 (Sept.) 1947.
10. Evans, F. T.; Gray, P. W. S.; Lehmann, H., and Silk, E.: Effect of pseudocholinesterase level on action of succinylcholine in man, Brit. M. J. 1: 136 (Jan. 17) 1953.
11. Foldes, F. F., and others: Synthetic muscle relaxants in anesthesia, J.A.M.A. 150: 1559 (Dec. 20) 1952.
12. Gabbard, J. G.; Roos, A.; Eastwood, D. E., and Burford, T. H.: Effect of ether anesthesia upon alveolar ventilation and acid-base balance in man, Ann. Surg. 136: 680 (Oct.) 1952.
13. Gray, T. C., and Rees, G. J.: Role of apnea in anesthesia for major surgery, Brit. M.J. 2: 891 (Oct. 25) 1952.
14. Hampton, L. J., and others: An Assessment of the Complications in 21,000 Cases and of Controlled Hypotension in Anesthesia, An Exhibit, 1952.
15. Johnstone, M.: Cyclopropane anesthesia and ventricular arrhythmias, Brit. Heart J. 12: 239 (July) 1950.
16. Lewis, F. J., and Taufic, M.: Closure of atrial septal defects with aid of hypothermia; experimental accomplishments and report of one successful case, Surgery 33: 52 (Jan.) 1953.
17. Liver necrosis after trilene anesthesia (Annotation) Lancet 1: 476 (April 8) 1944.
18. Maier, H. C.; Rich, G. W., and Eichen, S.: Clinical significance of respiratory acidosis during operations, Ann. Surg. 134: 653 (Oct.) 1951.
19. Miller, F. A., and others: Respiratory acidosis: its relationship to cardiac function and other physiologic mechanisms, Surgery 32: 171 (Aug.) 1952.
20. Moyer, C. A., and Beecher, H. K.: Effects of barbiturate anesthesia (evipal and pentothal sodium) upon integration of respiratory control mechanisms. Study directed toward improvement of methods for preclinical evaluation of anesthetic agents, J. Clin. Investigation 21: 429 (July) 1942.
21. Muller, W. H.: Personal communication to Dr. Henry Swan.
22. Mustard, W. T.; Chute, A. L., and Simmons, E. H.: Further observations on experimental extracorporeal circulation, Surgery 32: 803 (Nov.) 1952.
23. Orton, R. H.: Carbon dioxide accumulation, Anaesthesia 7: 211 (Oct.) 1952.
24. Southworth, J. L.; Peirce, E. C. II; Tyson, T., and Bowman, R. L.: Diaphragm pump for artificial circulation, Tyson-Bowman pump, A.M.A. Arch. Surg. 66: 53 (Jan.) 1953.
25. Stewart, B.D.; Virtue, R. W., and Swan, H.: Cardiac arrest and ventricular fibrillation. Experimental study in dogs with acute hypoxia and hypercapnia and in dogs with chronic hypoxia, Arch. Surg., 66, 703 (June) 1953.
26. Stormont, R. T.; Hathaway, H. R.; Shideman, F. E., and Seevers, M. H.: Acid-base balance during cyclopropane anesthesia, Anesthesiology 3: 369 (July) 1942.
27. Swan, H.; Zeavin, E.; Holmes, J. H., and Montgomery, V.: Cessation of circulation in general hypothermia. I. Physiologic changes and their control, Ann. Surg., to be published.
28. Taylor, F. H., and Roos, A.: Disturbances in acid-base balance during ether anesthesia, with special reference to changes occurring during thoracic surgery, J. Thoracic Surg. 20: 289 (Aug.) 1950.
29. Waters, R. M.; Orth, O. S., and Gillespie, N. A.: Trichloroethylene anesthesia and cardiac rhythm, Anesthesiology 4: 1 (Jan.) 1943.
30. Wesolowski, S. A., and Welch, C. S.: Experimental maintenance of circulation by mechanical pumps, Surgery 31: 769 (May) 1952.
31. Young, W. G., Jr.; Sealy, W. C.; Harris, J. S., and Botwin, A.: Effects of hypercapnia and hypoxia on response of heart to vagal stimulation, Surg., Gynec. & Obst. 93: 51 (July) 1951.
32. Zeavin, I.; Virtue, R. W., and Swan, H.: Cessation of circulation in general hypothermia. II. Anesthetic management, Anesthesiology, to be published.

HIP FRACTURES IN GENERAL SURGERY

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This paper is based upon a study of the experience of a general surgeon in the treatment of 80 patients with fractures of the upper end of the femur between the head and trochanteric region, from May 1938 to February 1953. These patients were operated upon in two small general hospitals.

HISTORICAL

The treatment of hip fractures prior to 1902, when Whitman's[26] work was published, was unsatisfactory and union of intracapsular fractures was rare. This undoubtedly had some relation to the development of the roentgen ray which began in the latter part of the eighteen-nineties.

Nails, screws, and pegs were used for internal fixation by a number of surgeons. Langenbeck used the method in 1850. J. Nicolaysen[15] described a nailing technic in 1897. Thomas[21] in 1921, and Martin and King[21] in 1922, used steel-wood screws which were not too successful. In 1921 Dilbet and Basset[3] reported fixation by means of a screw with four successful results out of 35 cases.

Whitman made a great contribution to the treatment of hip fractures by his careful study of roentgenograms and anatomic specimens which resulted in his method of reduction by abduction, extension, and internal rotation and fixation in a plaster spica. His percentage of union was estimated at 40. This was a great improvement over past results but leaves much to be desired.

Smith-Petersen[20] revived and popularized internal fixation by his invention of the three-flanged nail in 1922. This was the ideal instrument for internal fixation. The publication of his work in 1931 was the beginning of a new era in the surgical treatment of hip fractures. His successful work was made possible by that of Venable,[21] Stuck,[21] and others about the same time, by their discovery of vitallium, stainless steel, and other inert nonionizable metals which have little or no reaction on bone or soft tissues. Schilling[4] and Johannson[5] both used guide wires and modified the nail by cannulating it. The nail commonly used today is probably Johannson's modification, and the extra-articular method of nailing generally used today was worked out and improved upon by many men —Schilling, Johannson, Bohler, Westcott,[25] and others. Numerous pins and screws have been recommended, such as those of Moore, Knowles, Henderson, Moreira and others; but the cannulated Smith-Petersen nail has withstood the test of 25 years and, in the words of Watson-Jones,[24] "is one of the greatest surgical advances of this generation."

SELECTION OF PATIENTS

Few patients with hip fractures should be denied operation. Age is no contra-indication. In many series of cases—and in this series—patients 90 years of age and older have survived the operation and lived in comfort for some time. Because of the comfort, freedom from pain, and ease of turning the patient afterward, as well as the minimum risk involved, nailing is indicated for all patients, regardless of age, who have hip fractures, except the acutely ill and moribund patient. This statement is, of course, predicated upon the principle that one will reduce and nail the fracture as though the fracture would heal by bony union and the patient would live indefinitely. It should be understood that this statement includes impacted or nondisplaced fractures. This is the opinion of Cleveland and Bailey,[8] Speed and Smith,[21] Neer,[14] and many others. One case in this series convinced me of the necessity of nailing impacted fractures. One of the early cases reported in detail by me[4] in 1942 was not nailed, but was put in plaster for four weeks at the end of which time the fracture was found to be unimpacted and displaced. It was nailed at once and the man is alive and well today, 11 years later. He is 85 years of age and has bony union and no disability.

TIME FOR OPERATION

Patients in this series have been operated upon from 24 hours to one month after their fractures occurred. It is not thought that hip nailing is an emergency operation, but operation within 24 or 48 hours should be done unless a disease, such as diabetes or heart failure contraindicates it. Badgley's[2] results with non-union impresses one with the need for early operation. It has been shown by many that mortality rates and complications increase when operation is delayed over four or five days.

STEPS IN TREATMENT

The patients in the cases here discussed were all operated upon in small general hospitals, with no interns and with no personnel trained in orthopedic surgery; therefore, the technic is as simple as possible.

No traction is used before or after operation, except in badly displaced trochanteric fractures, in which it is used preoperatively. Some immobilization is obtained by sandbags. Laboratory studies are made and blood is matched prior to operation. Blood is used during operation in the majority of cases. A Foley catheter is placed in the bladder in the elderly patients, either before or just after operation.

Neck fractures and basilar extracapsular fractures without much displacement are operated upon on the roentgen ray table in the roentgenology department (fig. 1). All displaced trochanteric fractures are operated upon on the Hawley table with both feet and legs fixed firmly. The portable roentgen ray unit is used. For neck fractures, fixation or mild counter traction is obtained, as shown in the photograph, by placing a long folded sheet in the perineum, tied to a rope and fastened to a door hinge or water pipe over the patient's shoulder on the side opposite the fracture in line with the traction on the fractured hip. A lead

Fig. 1. Set-up for nailing fractures of neck of femur. Roentgen ray table used as operating table. Counter traction by sheet and rope fastened to door hinge. Leg held in position by assistant. Roentgen ray tube and film in position for lateral exposure.

marker, $\frac{1}{2}$ inch square, is placed slightly below the center of a line between the anterosuperior spine and the pubic spine, over the head of the femur. This is kept visible in the operative field.

Anesthesia. Almost every type of anesthetic has been used in this series, including local, spinal, ether, cycloprane, nitrous oxide and sodium pentothal. At present, the choice is nitrous oxide with a high concentration of oxygen, frequently preceded or supplemented with sodium pentothal.

The success or failure of operative treatment depends upon the accurate reduction of the fracture. It has been wisely stated that the bad results of nailing are the results of bad nailing. It is proved by hundreds of cases on record that nonunion and other complications are in proportion to poor or inaccurate reduction.

The extremity, with the knee in full extension, the leg in slight abduction, and 10 to 15 degrees internal rotation as estimated by the patella, is held by the foot and lower leg, by an assistant who is seated on a stool. This position brings the neck of the femur almost parallel with the table and an effort is made to maintain this position throughout the operation.

The thigh is prepared and draped; a short incision is made laterally from the greater trochanter downward, and the bone is exposed. A guide wire $\frac{3}{32}$ inch in diameter and 8 inches long is drilled into the center of the cortex, about 1 inch below the prominence of the trochanter, held parallel to the table or pointing slightly anteriorly and directed toward the lead marker over the head of the femur. It is drilled in a distance of 3 or 4 inches. A second guide wire is drilled in parallel and slightly proximal to the first.

An anteroposterior roentgenogram is made. The tube is rotated; brought underneath the opposite knee, and directed toward the neck and head of the femur. Radiographic film is held in an especially made holder recently obtained —which resembles a *popcorn popper*—so that the person holding the film is 3 feet or more away from it. It is pushed into the flank as far as possible parallel to the neck of the femur. Following the recommendation of Watson-Jones[24] we obtained a Lysholm grid which has greatly improved the lateral roentgenograms. The films are developed in one or two minutes in a superheated concentrated developing solution at a temperature of 70 to 72 degrees. They are overshot and underdeveloped because only the outline of the head and neck is desired.

According to J. S. Speed,[21] if reduction is not satisfactory in two attempts, one should open the hip joint and fix the fracture under direct vision. This advice would have been most welcome in the most difficult case in this series; a subcapital fracture in which 12 or 15 attempts at reduction were unsuccessful and the operation was stopped. Three weeks later an arthrotomy was done and the fragments were fixed under direct vision.

If reduction is satisfactory one guide wire is removed and a reamer is introduced over the remaining guide. A hole is reamed out of the cortex. An attempt it made to select a nail which goes within $\frac{1}{4}$ to $\frac{1}{2}$ inch of the articular surface of the head. The nail is driven in over the guide wire and the fragments are thoroughly impacted. Anteroposterior and lateral roentgenograms are made and the wound is closed in layers, without drainage. An effort is made to place the nail slightly in the inferior and posterior portion of the neck and head.

No traction or internal fixation is used after operation. The leg is placed upon a pillow. Compression elastic bandages are placed at once on both legs from toes to knee of every elderly or debilitated patient, in accordance with the teaching of Ochsner, just as is done in every patient of similar age having any type of major surgery. This is thought to reduce thrombo-embolic accidents to a minimum. The bandages are kept on until the patient is completely ambulatory.

Trochanteric fractures—except basilar fractures with little displacement— are operated upon on the fracture table. Both legs and feet are fixed in traction and are abducted to 135 to 150 degrees. Two portable roentgen-ray machines are placed in position and are not moved during the procedure. All patients receive blood in considerable quantity during operation, since the operation is bloody and shocking. The appliances used for fixation are listed in table III and shown in figure 2.

Although the Jewett appliance was used in the majority of cases, a Smith-Petersen nail with a Thornton, or Hopkins, or McLaughlin plate on the femoral shaft is easier to use. The guide shown in figure 2, set at an angle of 130 degrees to the shaft has been of considerable help with the Jewett appliance. The Moore-Blount blade plate has been abandoned because there have been cases in which the blade has cut out of the neck or head. The ideal sought in fixation here is a nail, which goes no closer to the articular surface than $\frac{1}{2}$ inch, and a plate of such length that two or three screws are below the fracture line on the shaft. In any case, in severely comminuted or displaced fractures, where there is a question of
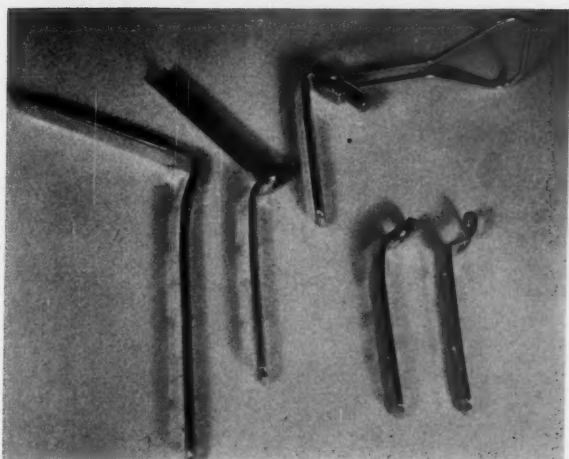
Fig. 2. Appliances for trochanteric fractures. Left to right: Moore-Blount, Jewett with guide at angle of 130 degrees, Lawson-Thornton, and Hopkins. The latter two for attachment to Smith-Petersen nail.

stability at the end of the operation, there is no hesitation in putting on a plaster spica which is left on for two to four weeks. Several bad experiences have taught me this lesson.

A recent article by Horwitz[11] describes a posterolateral approach for trochanteric fractures, with the patient in the prone position. The author claims easier reduction as well as fixation through this incision over the extracapsular portion of the neck and trochanteric region. Care must be taken to avoid injury to the posterior circumflex vessels. The method seems worthy of a trial.

POSTOPERATIVE CARE

All patients are given penicillin for five to seven days. Early ambulation is highly desirable, but surgical heroics are not practiced. As soon as a patient is oriented and fairly comfortable he is placed in a chair and is urged actively to move both legs. A detail just as important is to turn the patient on the uninjured side every day after operation and give careful attention to the back, as well as to all pressure points. This has been impressed upon me by disastrous bedsores. In bed, passive, gentle flexion of the knee and thigh is used daily until the patient can perform active motion. A stiff knee is a dreaded sequel to the injury.

After the wound is healed—in a week or 10 days—the patient is encouraged to walk in a walker rather than with crutches, touching the foot to the floor, but without weight bearing. Crutches may be used later if the patient is careful and is steady. No weight bearing is permitted until bony union is definite, as shown by roentgenogram, and if no vascular disturbance is seen. In the neck fractures in this series the time required for union was four to six months, and in

TABLE I

	Number of Cases
Neck	48
Trochanteric	32
	—
Total number of hips	80
Females	59
Males	21
1938 to 1942	21
1946 to 1953	59
Age distribution	39 to 90 years
Average age	70.7 years

the trochanteric group it was three to four months. In one subcapital fracture full weight bearing was not permitted for more than a year.

The distribution of fractures, as to types, sex, period of years, and age is shown in table I.

The average age of patients with trochanteric fractures is older, the shock and trauma are more severe, and the operative mortality should be higher.

Pauwels[16] in 1935 gave a classification of neck fractures based on the obliquity of the fracture line (fig. 3). His type 1, is an abduction fracture. It has an angle of 30 degrees or less to the horizontal, and usually heals. Here the shearing force promotes union. His type 2, or intermediate, with an obliquity of the fracture line of 30 to 50 degrees is subjected to severe shearing force and must be fixed firmly and remain fixed to obtain union. The type 3, or adduction fracture with a nearly vertical fracture line of 70 or more degrees—due to the shearing force— is much more likely to end in nonunion and should have the maximum in fixation. Pauwels' classification and its effect on prognosis has resulted in intensive study of the types of fractures and their treatment.
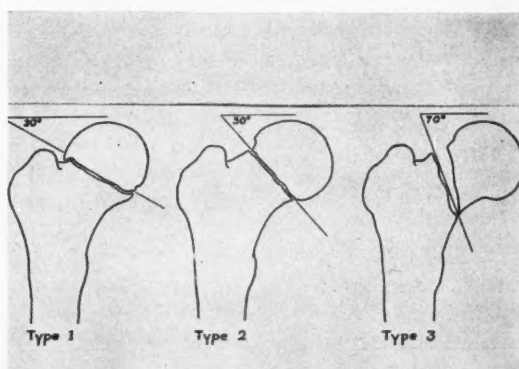

FIG. 3. Pauwels' classification according to degree of obliquity of fracture line to the horizontal.

TABLE II
Follow-up study

Follow-up: 10 years or more.. 8

5 years or more.. 11

3 years or more... 14

1 year or more... 28

Under 1 year... 19

Known deaths... 30

Unknown.. 3

Known living... 47

Neer[14] has given a very workable classification of trochanteric fractures, as follows: 1) nondisplaced or basal, 2) stable (which may be intertrochanteric or subtrochanteric), and 3) unstable (or comminuted displaced).

A follow-up study of patients is made in table II.

A concentrated effort has been made to obtain some type of report on all patients—preferably recent roentgenograms, or an examination, or a report from a relative or a report by letter. The condition of 8 patients is known at the end of 10 years. Roentgenograms of 4 of these patients show excellent results. The other 4 patients are known—through personal contact, letter, or by report of relatives—to be in good health and to have no disability. It is noted in table II that 40 per cent have been followed for more than three years and 75 per cent have been followed for more than one year.

TYPES OF INTERNAL FIXATION

The Smith-Petersen nail was used on all neck fractures and is most satisfactory.

Trochanteric fractures have presented the greatest problem, notwithstanding the old argument that they always heal. I used fixation by hip spica for many years. This was followed by the Roger Anderson or Carl Jones well-leg traction apparatus. In 1937 Thornton[22] devised a plate which is attached to a Smith-Petersen nail. Other devices which appeared were Moore pins, Knowles pins, Compere pins, the Neufield blade plate, the Jewett appliance, the Moore-Blount blade plate, Moe plate, the McLaughlin, and Hopkins plates. My preference of material is SMO stainless steel, largely because of the price. The cost constitutes my only objection to the McLaughlin appliance. Table III shows the appliances used in this series.

TABLE III
Types of internal fixation

	Neck	Trochanteric
Smith-Petersen nail.............................	48	2
Moore or Compere pins...........................		2
Smith-Petersen with Thornton plate..............		8
Moore Blount blade plate........................		4
Smith-Petersen Jewett appliance.................		14
Smith-Petersen with McLaughlin plate............		1

TABLE IV

Mortality

Age (yr.)	No. of Hips	Deaths	Per Cent Mortality
Under 40	1	0	
40 to 59	9	0	
60 to 69	21	0	
70 to 79	32	3	9.3
80 to 90	17	2	11.7
Total	80	5	6.25
1938 to 1942		0	
1946 to March 1950		5	
1950 to March 1953		0 (27 cases)	

Number of postoperative days before death	1 to 17
Average postoperative days before death	6½
Of five postoperative deaths	4 neck
	1 trochanter

MORTALITY

The mortality rate and age distribution is shown in table IV.

In most of reported series studied the mortality rate in the age group of 60 years or below was very low. Above 70 years it rises considerably and in proportion to age. In this series death occurring within approximately two weeks was listed as a surgical death. One patient included, died 17 days after operation. Several patients remained in the hospital many months and died there, but they were not considered surgical deaths. Some showed union of the fracture before death. This series differs from many, in that four of the five deaths were in patients having intracapsular fractures. Only 1 had a trochanteric fracture. This is contrary to the average mortality figures as will be seen in the table of comparative series. The causes of death were the usual causes in this age group: cardiovascular failure, pulmonary embolism, diabetes with complications, and one death from the anesthetic.

The recovery of elderly patients with displaced, comminuted trochanteric fractures—from the severe trauma to tissues of an operation lasting several hours—never ceases to amaze me, even with excellent anesthesia and adequate quantities of blood.

COMPLICATIONS AND DISABILITY

Under this heading a number of conditions are listed (table V).

In 2 patients it was discovered in a week or 10 days after the operation that the nail had slipped out for some distance. One was a basilar fracture and the other a mid-cervical fracture. Both were promptly renailed and bony union was obtained.

One case was a displaced subcapital fracture in a thin woman who was 53

TABLE V

Complications and disability

Number of Cases

1. Reoperations
   Due to slipping of nail 2
   Due to inability to reduce fracture (this case required arthrotomy). . . . 1
2. Aseptic necrosis 1 (1.25%)
3. Nonunion
   Trochanteric 2 (2.5%)
   Neck 0
4. Compound trochanteric fracture resulted in osteomyelitis, nonunion
       and amputation 1
5. Infection, postoperative (of short duration and of soft tissues only). . . 2
6. Shortening and varus deformity (cases of trochanteric fractures moder-
       ately crippled due to shortening of 2 inches or more caused by blade
       or nail slipping out of neck or head or absorption of neck; all have
       bony union) 7
7. Protrusion of nail through head, which Cleveland calls overdrive. One
       case discovered by roentgenogram two and a half years after opera-
       tion 1

years of age. Numerous unsuccessful attempts were made to obtain a satisfactory closed reduction. The operation was terminated and three weeks later an arthrotomy was done. Even under direct vision a reduction was obtained, which was far from perfect. The nail was driven in until it was just under the articular surface. A plaster hip spica was applied and was left on for two months. This patient has been carefully followed for 28 months. It can be readily seen that bony union is present. The patient walks without support, without pain, and with a good range of motion but with some limp.

Aseptic or avascular necrosis is defined by Badgley[2] as "death of part or the whole of the head of the femur due to vascular damage, primarily of the posterior and anterior circumflex vessels." The blood supply of the ligamentum teres may be injured similarly. Death of bone takes place in a matter of hours, but clinical evidence is not manifest for two or three months and it may be be seen for 18 months. Density is the chief diagnostic feature until collapse of the head is seen. It is frequently not seen until after bony union has taken place.

Watson-Jones[24] states that: subcapital fractures above the site of the vascular entry inevitably, transcervical fractures frequently, and basal fractures never undergo avascular necrosis. He further states that it might not be the result of the original injury, but can be produced by vascular damage from manipulation or open reduction, cutting the capsule and interfering with the blood supply. The frequency of this condition is reported to be distressingly high. It occurs both in fractures treated by external and internal fixation. It practically never occurs in trochanteric fractures. The per cent ranges from 10 to 40 or 50. It is much more common where there is nonunion. In the prevention of this condition Badgley[2] emphasizes gentle handling in transport and little or no manipulation in reduction but preferably traction. He also advises against open operation if it is possible to avoid it, and no weight bearing for six months or longer. If the condition does

Fig. 4. Woman aged 67. Pauwels type 2 fracture of neck

not regress the treatment is removal of the *dead head* and some type of arthroplasty or the use of one of the newer prostheses.

The 1 patient in this series is typical of avascular necrosis. A thin frail woman of 67 years had a fracture of the right upper neck region of the Pauwels type 2 variety. It was nailed without difficulty. In six months there was solid bony union and at the same time increased density and slight flattening of the head were noticed. In 11 months there was progression of the condition, although she was using crutches without weight bearing. In two years the head was collapsed, with no hope of recovery. The nail has been removed; the condition is worse but the patient refuses further surgery (figs. 4, 5 and 6).

There are only seven possible neck fractures—including those who died postoperatively or within one year—who might have developed aseptic necrosis if they had lived.
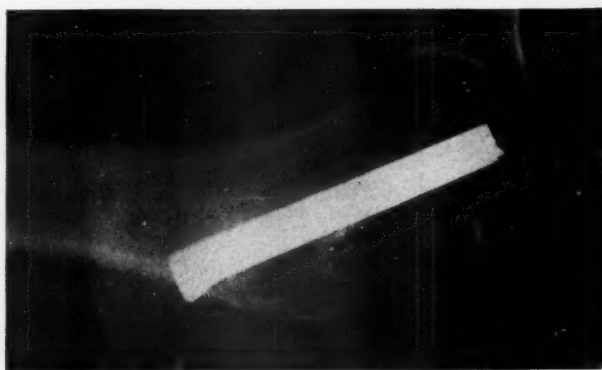

Fig. 5. Same as figure 4, after nailing

Nonunion. There were no cases of nonunion in the neck fractures. If all 7 patients had lived long enough to develop nonunion the total per cent would have been 10 or 11, since there were 2 cases of nonunion in the trochanteric group. This also is not usual. One of these was a compound fracture in a mal-nourished tabetic male, which finally resulted—after osteomyelitis and nonunion—in a hip joint disarticulaton. The other was really a refracture of a subtro-chanteric fracture which was thought to have healed and appeared after the man had done manual labor for six months.

Fig. 6. Same as figure 5. Two years postoperative. Advanced aseptic necrosis

Nonunion is a fairly common complication of neck fractures but practically never seen in those of the trochanter. Internal fixation has reduced the incidence from 60 per cent with the Whitman abduction plaster cast to from 10 to 20 per cent in most series of cases. It usually occurs with a few months but may occur up to a year or longer. Accurate reduction and correct nailing have a direct influence on nonunion. In Boyd's[7] series 72 per cent of the cases of union had ex-cellent reduction and nailing, and 28 per cent of the cases of fair, or poor reduc-tion and nailing showed union. The per cent of nonunion is higher in Pauwels' type 2 and 3 fractures. About 50 per cent of cases of nonunion have a poor reduc-tion; inadequate fixation; or early removal of the nail.

Because of the high incidence of nonunion in the vertical type of fractures, many attempts have been made to improve fixation and to insure union. King,[12]

Fig. 7. Woman aged 73. Trochanteric fracture of left hip treated with Smith-Petersen nail and Thornton plate. Three years later trochanteric fracture of right hip treated with Jewett appliance. Lived three months after last operation and obtained union.

in 1939, advocated bone graft in addition to the Smith-Petersen nail. Fischer and Leatherman[10] recently have reported 66 cases in which they have used two fibula grafts driven in above and below a Smith-Petersen nail. Their results have been most encouraging.

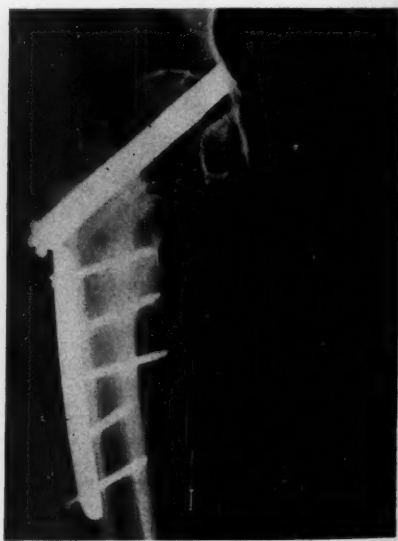Fig. 8   Fig. 9

Fig. 8. Woman aged 55. Roentgenogram taken day after operation
Fig. 9. Same as figure 8. Three years postoperative. Protrusion of nail through acetabulum from overdrive.

Ankner and Nelson[1] advise primary wedge-type osteotomy utilizing a modified Moe plate and long hip screws for fixation. They report 75 per cent bony union and believe the functional results are better. Other methods are constnatly being advocated to lower the per cent of nonunion.

Infections have been of little consequence in this series.

There has been far more deformity, shortening, coxa vara, and arthritis in the trochanteric group than in the neck group. More difficulty was encountered with the Moore-Blount appliance than in the others. One patient sustained a trochanteric fracture on one side which was treated by a nail and Thornton plate with complete recovery; followed three years later by a similar fracture of the other hip which was treated with the Jewett appliance and made a similar recovery with union (fig. 7).

TABLE VI

Comparative End Results in Percentages

Author	Mortality		Aseptic Necrosis, Neck	Nonunion, Neck
	Neck	Tro-chanter		
Whitman				60
Watson-Jones			Frequent—in neck, never in trochanter	10–25
Cleveland and Associates (1948)		12.6		
Fracture Committee Acad. O. S.	8.5		40–26.6	30
Badgley				10 in 48 hrs. 35 in 2 wks.
Boyd and George (1948)	7	13	32.6 (union) 60 (nonunion)	15.3
Neer (1951)	7–9	23	45	19
Cleveland and Bailey (1950)	7.2		21.3 (15 in last 59)	20.3 (internal fixation) (77 plaster of paris)
Speed and Boyd	9	13.7		
Horwitz (40 patients)	5		Rare	
Ankner and Nelson (45 patients)	6.7		9.3	20
Semb[19] (117 patients)			8.0	10
Lewis, Boutelle and Roberts[13] (152 patients)	7.8		0.75	25.5
Ryan and Gosslee[18]		3* 29†		
Baughman (80 patients) (1953)	6.25 (includes both types)		1.25	2.5 (2 cases. Both were subtrochanteric)

* Surgical.
† Conservative.

The same experience occurred in this series which surprised Cleveland[9] in his series—that is: protrusion of the nail through the head and into the acetabulum from overdrive. In a check-up roentgenogram of a mentally defective patient three years after operation the condition seen in the roentgenograms (figs. 8 and 9) was discovered. The patient walked with a limp but did not complain of pain.

Comparative mortality rates and complications are shown in table VI. These results were obtained from a number of series reviewed. The results in my small series are not out of line with these.

SUMMARY AND CONCLUSIONS

A series of 80 hip fractures treated by internal fixation over a period of 15 years has been studied and reported. Forty-eight were fractures of the neck and 32 were of the trochanteric region.

The literature has been reviewed and other series compared with this one.

Forty per cent of the patients were followed for more than three years and 75 per cent for more than one year. Ten per cent or 8 patients followed for 10 or more years have good, or excellent, results.

The operative or fracture mortality rate from 1 to 17 days after operation was 6.25 per cent (5 cases). There have been no deaths in the last 27 patients operated upon. Four of the deaths were in neck fractures and one in a trochanteric fracture.

All of the neck fractures followed, obtained bony union and two trochanteric fractures resulted in nonunion.

Only one of the neck fractures showed avascular necrosis, beginning in six months and advanced in 12 months. It is rarely seen after 18 months.

The average age of patients was 70.7 years.

The best way to obtain uncomplicated union is by accurate, anatomic reduction and firm, sound, prolonged internal fixation, without weight bearing until union is seen by roentgenogram.

Difficulties found in trochanteric fractures were: coxa vara deformity, shortening, arthritis, limp, and overdrive causing protrusion of nail into hip joint.

All fractures—including impacted fractures—should be nailed, regardless of age of the patient, except in the acutely ill and the moribund.

The internal fixation material of choice is the SMO stainless steel cannulated Smith-Petersen nail for neck fractures, and the nail and Thornton or McLaughlin plate, or the Jewett appliance for trochanteric fractures.

All fractures should be operated upon within 24 to 48 hours.

Neck fractures may be satisfactorily operated upon without traction and on a roentgen ray table.

Displaced trochanteric fractures should be operated upon in traction upon a fracture table.

The nailing of the subcapital, vertical fracture should be supplemented by bone grafting.

Adequate anteroposterior and lateral roentgenograms are indispensable.

Good general anesthesia and adequate blood transfusions during operation maintain a low mortality rate.

Patients should be out of bed as early as practicable but should not bear weight until union is seen on roentgenogram.

REFERENCES

1. Ankner, F. J., and Nelson, M. C.: Primary osteotomy for treatment of intracapsular fracture of neck of femur, Ann. Surg. *135:* 69 (Jan.) 1952.
2. Badgley, C. E., and Denham, R. H.: Aseptic necrosis of femoral head following fracture of hip, J. A. M. A. *137:* 1193 (July 31) 1948.
3. Basset, A., and Dilbet, P.: Fractures du Col de Femur, Paris, 1921.
4. Baughman, B. B.: Nailing of fractures of neck of femur, Kentucky M.J. *40:* 303 (Aug.) 1942.
5. Böhler, L.: Treatment of Fractures ed. 4, Baltimore, William Wood & Company, 1935.
6. Boyd, H. B., and George, I. L.: Complications of fractures of neck of femur, J. Bone & Joint Surg. *29:* 13 (Jan.) 1947.
7. Boyd, H. B., and George, I. L.: Fractures of hip; results following treatment, J. A. M. A. *137:* 1196 (July 31) 1948.
8. Cleveland, M., and Bailey, W. L.: End result study of intracapsular fracture of neck of femur, Surg., Gynec. & Obst. *90:* 393 (April) 1950.
9. Cleveland, M., Bosworth, D. M., and Thompson, F. R.: Management of trochanteric fracture of femur, J. A. M. A. *137:* 1186 (July 31) 1948.
10. Fischer, K. A., and Leatherman, K. D.: Smith-Petersen nailing with fibular bone grafts as primary treatment for fractures of neck of femur, Kentucky M.J. *50:* 146 (April) 1952.
11. Horwitz, T.: Posterolateral approach in surgical management of basilar neck, intertrochanteric and subtrochanteric fractures of femur, Surg., Gynec. & Obst. *95:* 45 (July) 1952.
12. King, T.: Closed operation for intracapsular fractures of neck of femur, Brit. J. Surg. *26:* 721 (April) 1939.
13. Lewis, K. M., Boutelle, W. E., and Roberts, M. A.: Intracapsular fracture of neck of femur. Observations on one hundred and fifty-two patients treated by internal fixation with Smith-Petersen nail, Ann. Surg. *131:* 376 (March) 1950.
14. Neer, C. S. II: Symposium on orthopedic surgery; surgical treatment of fractured hip. S. Clin. North America *31:* 499 (April) 1951.
15. Nicolaysen, J.: Lidt om Diagnosen og Behandlingen af Fr. colli femoris, Nord. med., Ark., Stockholm n.f., viii, Festbd. Axel Key, no. 16, 1–19, 1897.
16. Pauwels, F.: Der Schenkenhalsbruch, ein Mechanisches Problem, Ztschr. f. orthop. Chir. *63:* 32, 1935.
17. Report of the Fracture Committee of the American Academy of Orthopedic Surgeons; Treatment of fractures of neck of femur, J. Bone & Joint Surg. *23:* 386 (April) 1941.
18. Ryan, J. J., and Gosslee, J. M.: Intertrochanteric fractures of hip; six year study of two hundred and thirteen cases, Ann. Surg. *134:* 822 (Nov.) 1951.
19. Semb, C.: Technic of nailing of fractures of neck of femur, Surgery *4:* 321 (Sept.) 1938.
20. Smith-Petersen, M. N., Cave, E. F., and Van Gorder, G. W.: Intracapsular fractures of neck of femur; treatment by internal fixation, Arch. Surg. *23:* 715 (Nov.) 1931.
21. Speed, J. S., and Smith, H., editors: Campbell's Operative Orthopedics, ed. 2, St. Louis, C. V. Mosby Company, 1949.
22. Thornton, L.: Treatment of trochanteric fractures: two new methods, Piedmont Hosp. Bull. (Atlanta, Ga.) *10:* 21, 1937.
23. Van Gorder, G. W.: Treatment of acute fractures of neck of femur, J. A. M. A. *137:* 1181 (July 31) 1948.
24. Watson-Jones, R.: Fractures and Joint Injuries, ed. 3, Baltimore, Williams & Wilkins Company, 1944.
25. Wescott, H. H.: Method for internal fixation of transcervical fractures of femur, South. Med. & Surg. *96:* 458 (Sept.) 1934.
26. Whitman, R.: New method of treatment for fracture of neck of femur, Tr. Am. Orthopaedic A. *15:* 338, 1902.

EDITORIAL

A FORMULA FOR INTERESTING AND INSTRUCTIVE HOSPITAL STAFF MEETINGS

Practically every surgeon has some hospital connection that requires him to attend hospital staff meetings. It is with the idea of presenting a formula for interesting and instructive hospital staff meetings that this editorial is written.

This formula was not born full-grown but has evolved after many years of observing and participating in staff meetings in a small town hospital. The factors that have contributed to time consuming and futile discussions have been minimized under this formula by the simple procedure of priority. Instead of taking up the matter of unfinished or new business early in the evening the following technic is used.

The meeting is opened promptly on time. The first item at every monthly meeting is a short biographical sketch of someone who has made a worthwhile contribution in the field of medicine. It is requested that the writer of the sketch give brief biographical data and then attempt to appraise the contribution of the subject. In recent months very interesting sketches have been presented on James C. DaCosta, Sir James McKenzie, Sir William Osler, Hugh Young and Lord Berkeley Moynihan. In addition an interesting Review of the Introduction of Anesthesia was presented. This particular part of the program is limited to about 15 or 20 minutes.

The main theme of the evening then is presented by one or more members of the staff. The pattern of this is left to the discretion of the speakers. It may follow the conventional pattern of a Clinico-Pathologic Conference. It is requested that the presentation be distinctly clinical. Patients in the hospital or histories of patients in the hospital or histories of former patients are regularly used. After the subject has been presented, if pathologic specimens are available, the pathologist projects on a screen microscopic slides showing the pathology under discussion. This second part of the program is limited to about 45 minutes.

The program that has been presented has taken about an hour. The minutes of the last meeting are then read; unfinished business is acted upon; committee reports are heard and any new business is presented. We have found that the tendency is for the members to be less inclined to prolong the discussion in the closing part of the program than they are in the early portion. Effort is made to have important matters presented to the executive committee of the staff for discussion and recommendation. The executive committee of the staff meets weekly and at the monthly meetings of the entire staff they have recommendations to make which are usually accepted without prolonged discussion.

The members of our staff have been pleased with this type of meeting. They think it gives variety and balance to the program. We believe that it helps to develop, in the busy doctor, an appreciation of the continuity of medicine. We are very sure that the sequence tends to decrease prolonged and often profitless discussion of matters pertaining to nursing service or administration policy.

But the greatest value of such a program followed month after month accrues to the staff members themselves in helping them develop an appreciation of the continuity of medicine. *Possibly nothing would help public relations more than for all doctors to become imbued with the inherent dignity of our profession.*

We know, of course, that the above formula is no *Open Sesame* to successful staff meetings but at the Anderson Memorial Hospital we are finding this year that it is a workable formula. If any hospital staff is having problems with their staff meetings we recommend a trial of some such formula.

> J. R. YOUNG, M.D.
> *Anderson, S. C.*

BOOK REVIEWS

Nash's Surgical Physiology. A Second Edition Revised and Edited by BRIAN BLADES, M.D., Professor of Surgery, The George Washington University, School of Medicine, with the collaboration of EDWARD J. BEATTIE, JR., M.D., LEON GERBER, M.D., WALTER H. GERWIG, JR., M.D., ERNEST A. GOULD, M.D., ALEC HORWITZ, M.D., VINCENT M. IOVINE, M.D., GORDON LETTERMAN, M.D., WILLIAM S. McCUNE, M.D., WILLIAM C. MELOY, M.D., HUGO V. RIZZOLI, M.D. and JACOB J. WEINSTEIN, M.D., all of the George Washington University School of Medicine. Charles C Thomas, Springfield, Illinois, 1953. $12.50.

The first edition of Nash's Surgical Physiology was published in 1942. After 11 years the second edition has now appeared. During these 11 years much has been added to our knowledge of physiology as applied to surgery. This knowledge has been brought up to date in the new Nash's Surgical Physiology edited by Brian Blades. What is of value in the old book has been retained in this new edition. A glance through the extensive bibliography which appear at the ends of the various chapters attests to the great extent to which knowledge of physiology has advanced since 1942.

In the first edition Nash stated that "Operative technique is only the apex of the great pyramid of surgical knowledge; it is the broad and heavy base that is most needed and most difficult to construct." Nothing should be more stimulating to the modern surgeon, especially the young developing surgeon, than a study of this broad and heavy base of the pyramid which is embodied in this work on Surgical Physiology.

Some of the notable additions in the new book are, the study of intracardiac pressures by catheterization of the heart; applied physiology in the newer operations upon the heart and vascular system; recent concepts of surgical shock; physiology of burns and tissue repair; physiology of body fluids, acid base balance, and the role of the electrolytes in the treatment of surgical patients; adrenal physiology and its relation to treatment; effects of vagotomy on the physiology of the stomach; sympathectomy in peripheral vascular diseases; further study of physiologic changes in arterio-venous fistulas; the newer concepts of hypertension; and important additions to the physiology of the digestive system.

The text is supplemented by excellent explanatory drawings and tables.

This book emphasizes again that the surgeon must first be a physician. The art of surgery is not enough. Before and after operation patients must be studied and treated from the standpoints of physiology, chemistry and biology.

In this volume of Nash's Surgical Physiology the essential facts about normal and abnormal physiology are presented in a concise and lucid manner. To the teacher of surgery it should be a text. To all interested in keeping abreast of the times in the study of Surgery, from the medical student to the experienced practitioner, it should always be at hand for reference.

THOMAS G. ORR, M.D.

An Atlas of Pelvic Operations. By LANGDON PARSONS, M.D., Professor of Gynecology, Boston University School of Medicine; Chief, Department of Gynecology, Massachusetts Memorial Hospital; Gynecologist, Palmer Memorial Hospital; Gynecologist, Massachusetts Department of Public Health, Hospital for Cancer at Pondville, Massachusetts; and HOWARD ULFELDER, M.D., Assistant Surgeon, Massachusetts General Hospital; Gynecologist, Massachusetts Department of Public Health, Hospital for Cancer at Pondville, Massachusetts.

For the first time in recent years an excellent atlas of pelvic operations appears in print. The atlas contains 229 pages which mainly consist of superb drawings, illustrating the technical details of pelvic surgical procedures. The amount of descriptive writing accompanying each drawing is brief but adequate. As the authors state in the preface, this atlas covers only the operative technics which are "standard in institutions where we work."

The text is divided into three sections: (1) abdominal operations; (2) vaginal and perineal operations; (3) operations for malignant diseases. Included in these sections are surgical procedures involving the intestines, urinary tract and the abdominal wall, which should be understood by all surgeons doing pelvic operations. Especially noteworthy is the radical vulvectomy and radical groin dissection procedures which heretofore have been poorly understood by many. In an atlas of pelvic operations, the surgical technic of Cesarean section should be included.

The size of an atlas is definitely a disadvantage in one's library. This atlas will have its greatest usefulness as a review and a reference for the surgeon operating in this field. It is the reviewer's opinion that this atlas should not be used for extending one's field of operation in which the surgeon has had no previous experience.

C. A. HUNTER, M.D.

The Anatomy and Surgery of Hernia. By LEO M. ZIMMERMAN, M.D., Professor of Surgery and Co-Chairman of the Department of Surgery, Chicago Medical School. Attending Surgeon Michael Reese, Cook County, and Chicago Memorial Hospitals; and BERRY J. ANSON, Ph.D., Professor of Anatomy, Northwestern University Medical School. Member of the Attending Staff, Passabant Memorial Hospital. The Williams & Wilkins Company, Baltimore, 1953. $10.00.

This monograph on hernia fulfills a need for a volume which gathers the newer information published in the last few years, without attempting to be encyclopedic in scope. This is the authors' intention as expressed in their preface. They advocate particularly the performance of herniorrhaphy with relevance of the operative technic to the anatomical problem involved, and condemn the 'rote' type of surgery, in which too often a standardized procedure is done regardless of the pathology present. To augment this fundamental approach to the problem, their third, fourth, and fifth chapters are devoted exclusively to the anatomy of hernia, with numerous excellent illustrations, many of which have already appeared in Anson's Atlas of Human Anatomy.

The organization of the material under chapter headings is good, and specific points of information are readily accessible. The chapters on the history of hernia and their primitive management, and the medicolegal aspects of hernia command special interest, and add to the value of the monograph.

Some of the operative technics advocated may be challenged by different authorities, but the book has much worth to both the resident in surgery and the practitioner group, particularly the former. To the general practitioner, who is called upon to do an occasional herniorrhaphy, the volume is a very useful piece of armamentarium.

THOMAS G. ORR, JR., M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Diseases of the Retina. By HERMAN ELWYN, M.D., Senior Assistant Surgeon, New York Eye and Ear Infirmary. Second edition, revised and augmented with 243 illustrations of which 20 are in color. The Blakiston Company, Inc., New York, Toronto. $12.00.

Pictorial Introduction to Neurological Surgery. By G. F. ROWBOTHAM and D. P. HAMMERSLEY. E. & S. Livingstone, Ltd., Edinburgh and London. 1953. The Williams & Wilkins Co., Baltimore. $4.50.